VOLUME 13
NUMBER 6
JUNE, 1960

ORAL SURGERY
ORAL MEDICINE
and ORAL PATHOLOGY

OPERATIVE ORAL SURGERY

EXPERIMENTAL USE OF PROTEOLYTIC ENZYMES IN THE TREATMENT OF ORAL AND ASSOCIATED LESIONS

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NECROSIS or tissue death is a problem which the dental and medical practitioner faces often. This pathologic process involves the body in a situation of stress, namely, that of physiologic removal of the necrotic tissue and, of the inflammatory elements and fluid that accumulate due to the dead tissue. This constant struggle, which is basic to the maintenance of homeostasis, is the subject of this discussion.

Dead or dying cells release proteases which begin to split up the cellular proteins. The leukocytes also help by producing proteolytic enzymes, by digesting the debris, and by phagocytosis.

In some cases in which the dead cells produce no proteolytic ferments or the latter are present in insufficient amounts and the material is not chemotactic, according to Menkin's hypothesis, the body protects itself by attempting to wall off or isolate the insulting area.[1]

The presence of necrotic tissue and its products constitutes a threat to contiguous normal viable tissue, and thus it behooves us to aid the body in the rapid removal of these dead elements.

There are various methods by which the practitioner can speed this removal. Surgical débridement is an excellent aid. This, however, has drawbacks which limit its everyday use. Chemicals, such as urea, and thiourea as well as those which release chlorine, have been used. One of the better agents used to date is trypsin.

Trypsin, a proteolytic enzyme derived from mammalian pancreas, is being successfully used in many fields of dentistry and medicine[2] for selective physiologic débridement of necrotic tissue. Trypsin, in a highly purified crystalline

Thesis submitted in partial fulfillment of the requirements for the master's degree, Graduate School of Medicine, University of Pennsylvania.

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form, has been used clinically for the removal of necrotic tissue in varicose ulcers, open infected wounds, diabetic gangrene, soft-tissue abscesses, sinuses, and fistulas, burns covered with eschars, osteomyelitis, and hematomas, as well as in endodontics and periodontics. Because of its vigorous fibrinolytic action, trypsin was successfully used as an aerosol for adjunctive therapy in such disorders as bronchial asthma, bronchitis, and bronchiectasis, which are marked by hypersecretion and fibromucinous sputum.[3] More recently trypsin has been successfully used parenterally.

The enzymatic digestion of necrotic tissue is not a new idea; rather, it dates back to the middle of the eighteenth century and Dr. John Hunter (1728-1793). Hunter was the first surgeon to demonstrate the digestion of dead tissue by animal proteolytic enzymes.[4] In 1783, Spallanzani reported that the gastric juice of hawks liquefied meat.[5]

The oldest recorded medical histories contain evidence of the invasion of infected wounds by maggots, and this has been reported as beneficial. It is interesting to note that Miller discovered that trypsin was the enzyme system usually found in the excreta of maggots.[6]

Since the eighteenth century numerous proteolytic enzymes, of both plant and animal varieties, have been discovered, but these were given little attention since nothing was done to apply this knowledge toward the treatment of dental or medical lesions in man.

Tillett[7] and others have been investigating proteolytic enzymes of streptococcal origin for many years. These enzymes, streptokinase and streptodornase, are often together although they act differently. Streptokinase activates a factor in human serum, plasminogen, which splits fibrin into its formative elements, polypeptides.[8, 9] Streptodornase acts to split the nucleoprotein molecules, resulting in decreased viscosity of purulent exudates.[10] Streptodornase has no effect on the nuclei of living cells.[9]

Recently Miller and others have given intramuscular injections of streptokinase for the control of edema and infections. Parenteral streptokinase should be used only as an adjunct to antibacterial or antibiotic therapy and should not be given to patients with known defects in their clotting mechanisms. This This regime was used in cases of acute cellulitis, lymphangitis and lymphadenitis, acute purulent frontal sinusitis, multiple lacerations and abrasions, and edema following extraction of infected teeth. Most of the patients observed showed beneficial clinical effects after twenty-four hours of treatment with 5,000 units of streptokinase in 0.5 c.c. physiologic saline administered twice daily for at least six doses.[11]

Streptococcal proteolytic enzymes have certain properties which put them at a disadvantage, especially for topical application, when compared with crystalline trypsin. Streptococcal enzymes necessitate more exacting methods of storage and require higher concentrations and longer periods of contact for active lysis of tissues under treatment.[12] While desiccated streptokinase-streptodornase is stable at room temperature, it should be stored at 4° C. to maintain the most efficient potency. Streptokinase is inactivated by trypsin and possesses antigenic properties which may result in the formation of an

antikinase in the globulin fraction of the blood.[13] In addition, adequate circulation to the local area in question is necessary, since the action of streptokinase is limited by the amount of human serum factor present at the site.[13] For these reasons, streptokinase-streptodornase combinations are used less frequently by today's investigators; meanwhile, trypsin is gaining more and wider attention.

In 1949 Kurt Stucke,[14] a German investigator, reported the successful use of trypsin in enzymatic débridement of severe burns. Trypsin has since been used in the débridement of a great many suppurative and septic lesions all over the body.

In vitro, trypsin digests fibrin and clotted blood quickly and quantitatively. When applied in vivo to a purulent and necrotic lesion, trypsin rapidly lyses the fibrinous strands and coagulum on the surface of the lesion. This lysis takes place in a matter of minutes. It digests the slimy, viscid coating of desoxyribose nucleoprotein of the purulent exudate and then attacks any dead tissues in the lesion. There is an almost immediate outpouring of serum, which floats off the digested debris. The exudate becomes thin and serous. Bacterial rapidly decrease in number and finally may disappear completely. As treatment is continued and the exudate decreases or ceases, a clean, granulating pink surface is left.[3]

"Trypsin is innocuous to living tissue because the serum and the membranes of living cells contain a specific trypsin inhibitor and because there are several non-specific inhibitory substances which protect viable tissue from its proteolytic action."[15] Trypsin "aids the natural defense of the body to overcome the pathologic organisms present. Tryptar is neither directly bacteriostatic nor bactericidal but acts as a chemical aid to the defense mechanism of the body by its proteolytic action on necrotic cells and tissue. Thus, it removes the culture media for the chronically irritating organisms which are present."[2] Concomitantly, fresh viable leukocytes are brought to the area in large numbers, thus increasing the phagocytosis of the remaining bacteria.[4] This unique activity of trypsin without doubt cuts down on operating time and also reduces the number of postoperative visits required. To reduce a primary infection further or to prevent secondary invaders, an antibiotic or a combination of antibiotics may be mixed in with the solution of trypsin and buffer.[4, 16]

The enzyme trypsin is available commercially in capsules containing 250 mg. of powder. Certain general rules have been noted concerning the clinical use of topical trypsin. These rules should be observed if one is to obtain maximum successful results.[16] Proteolytic activity is optimal between pH 6.8 and 7.5,[4] the maximal activity being at pH 7.1. Therefore, when trypsin is used in cavities it should be employed in a solution of Sorensen's phosphate buffer. Prior to its use on a surface lesion, the pH of the surface should be tested. If it is found to be extremely alkaline or too acid, the trypsin should be used in a buffered solution and is best applied as a wet dressing. When a surface lesion is found to be within the optimal pH range, then the powdered trypsin crystals may be applied directly to the area. Tests for the local hydrogen ion concentration of the oral cavity may be made with nitrazine paper, which

gives a colorimetric index.[16] It is of interest to note that the oral cavity due to saliva, has a normal pH range of 6.0 to 7.9 and thus enhances the proteolytic activity of trypsin. Also, "there is evidence that normal individuals under 20 years of age and without dental defects secrete a saliva with a pH of between 7.0 and 7.2."[17] This is ideal for tryptic activity.

The second factor to be considered in the application of trypsin to a surface lesion is that of temperature. Any temperature below body temperature rapidly inactivates trypsin, as it does all other digestive enzymes.[17] Therefore, external heat should be utilized whenever surface dressings are applied. In the oral application of trypsin the thermal factor is near optimum and thus needs no special attention.

A third consideration is the time element. At room temperature a solution of trypsin loses 75 per cent of its enzymatic activity after three hours.[4, 16] Thus, the solution should be prepared just prior to the time of use, and for continuous activity any dressings used must be changed every three hours. There is an alternate method wherein tubes are incorporated in the dressing and fresh trypsin solution is instilled at three-hour intervals.

When trypsin is employed in a body cavity, a histamine-like reaction may take place with the first few treatments, due to continuous absorption.[18] This may be prevented by prior administration of 25 to 50 mg. of diphenhydramine (Benadryl) hydrochloride intravenously, or it may be eliminated by the oral administration of 50 mg. of Benadryl at three-hour intervals.[19] The only side reactions noted with the use of trypsin in body cavities have been a mild elevation of temperature and an increased pulse rate. When trypsin is used on the surface, the reactions encountered are in the form of a mild burning or stinging sensation. If necessary, the activity of trypsin may be stopped immediately by flushing the area with a sodium bicarbonate solution.[4]

Even more recently clinical investigators have used trypsin parenterally, and their findings are very encouraging. The difference between parenteral and topical uses of trypsin is considerable, mainly due to the quantities needed. Topical or aerosol administration must employ large amounts of trypsin, since the extent of substrate depolymerization is proportional to the quantity of enzyme introduced. The reaction differs also from the reaction to parenteral trypsin in that a trypsin-protein substrate reaction occurs.[20]

As for quantities required, an intramuscular trypsin preparation, such as Parenzyme, contains only 2.5 mg. of crystalline trypsin in sesame oil per 0.5 c.c. dose. The actual dosage schedules seem to differ with the various clinicians. Miller and co-workers report the best dosage to be about 0.5 mg. of trypsin dissolved in 1 c.c. of physiologic saline and given intramuscularly twice a day for three days.[21] Innerfield and associates[22] reported on the use of trypsin where there was evidence of inflammatory disease of the respiratory tract associated with viscid, sticky secretions. They successfully used a regime of one daily 0.5 c.c. intramuscular dose of Parenzyme (2.5 mg. of crystalline trypsin) plus one sublingual or buccal tablet every four to six hours. Each tablet also contained 2.5 mg. of crystalline trypsin.[22]

When trypsin is used parenterally, the minute amounts of enzyme per dose would seem to account for the low frequency and the mildness of side effects. About the only effects encountered and reported were a tingling of the tongue following administration of the sublingual tablets and some local tenderness following intramuscular administration.

There is no doubt that parenteral use of trypsin evolves a reversal of inflammation and edema. The exact mechanism of this reversal is not understood, but it is cogent to this discussion that current hypotheses concerning the mechanism be expressed. Some investigators believe that the dose of parenteral trypsin can be small because its physiologic action seems to be a triggering of the conversion of plasminogen, a precursor, into plasmin, the active enzyme. Thus, this small dose in some way initiates the activity of the patient's own proteolytic enzyme system. The result of this activity is an enzyme-protein complex of the *plasmin-protein substrate type.* This mechanism is considerably different from the *trypsin-protein substrate* reaction previously mentioned.[23, 24]

Other workers state that trypsin produces a reversal of inflammation and edema and explain it on the basis of its fibrinolytic action. That is, trypsin liquefies thrombi of fibrin in arteries, veins, and lymphatics as well as the fibrin transported via the edema fluids. This enhances circulation to the area and allows the natural forces and the antibacterial drugs to act more effectively at the site of trouble.[21] It is important to add here that due to this type of activity, which is actually an effective breakdown of normal body defenses and reactions, it is essential that an antibacterial drug be administered concomitantly. Also due to the possibility of hemorrhage, trypsin should not be given parenterally to patients with defects in their blood clotting mechanisms.

The reversal of inflammation described has been effectively demonstrated by many clinicians. Innerfield,[25] for example, reported on 141 patients. The results were reported under four headings: trauma, acute infections, thrombophlebitis, and upper respiratory disease. All four categories showed rapid improvement.

To date the clinical application of trypsin is still in its infancy, and in dentistry in particular it is little more than a pregnant idea. Thus, one of the aims of this article is to outline some of the possible dental aspects in which the new material may be brought to bear with great advantage to both patient and doctor.

It is my opinion that trypsin could be used to advantage in the treatment of lesions of the mouth and associated structures by previously outlined procedures. Specific indications for topical application might be such conditions as ulceromembranous stomatitis, ulcerative and desquamative stomatitis, necrotic gingivitis, dry sockets, necrotic wound sites, surgical sloughs, fistulas, and any of the stomatitides arising from blood dyscrasias, etc.

By way of example, one of the more difficult problems in oral surgery is the treatment and control of oral-antral openings, especially those complicated by infection. Cameron[26] has stated that the nasal sinuses will tolerate infection from the nasal cavity better than infection originating in the oral

cavity. Thus, it becomes the obligation of the oral surgeon to treat any oral-antral openings and to bring about the most rapid healing and closure possible. There are various surgical procedures for accomplishing this.

Thoma[27] has described four basic procedures for irrigation and drainage of the maxillary sinus. The incorporation of the use of trypsin and Sorensen's solution as a step in any one of the four treatments would enhance manyfold both the surgical success and the time factor of that success.

In all cases the dental cause must first be removed. In the case of the abscessed tooth, which is the primary cause of the maxillary sinus involvement, the tooth should be extracted and a trypsin solution could then be instilled into the sinus through the alveolus. This may be accomplished by using a soft rubber catheter. Surgical drainage may be effected by means of a trocar with a cannula, a Caldwell-Luc operation, or a modification thereof if necessary.

The use of the trocar is the simplest of the surgical procedures. "A trocar with a cannula is introduced through the nostril, inserted for about one inch beneath the inferior turbinate and pushed into the antrum through the thin bony partition. The trocar is removed, leaving the cannula in situ in order to evacuate the pus by suction."[27] This is usually followed by irrigation with normal saline solution, but by substituting a trypsin solution for the saline solution a more rapid and complete evacuation of the pus and dead material may be effected.

The Caldwell-Luc and Denker operations are more radical antrum procedures. In both operations two separate openings are made—one in the naso-antral wall for drainage and the other through the canine fossa or lateral antral wall to gain access. In both, the addition of trypsin for débridement would be highly beneficial.

The oral-antral fistula is another example of infection of the maxillary sinus resulting in failure of the healing process to close off the antrum. This process usually becomes associated with a chronic sinus infection, and often removal by a Caldwell-Luc operation is required prior to any plastic surgery. In this case, the addition of trypsin instillation to the surgical procedure would further ensure the removal of the chronic infection, leaving a smooth new granulating mucosa.

On the basis of the significant literature on enzymatic débridement of human tissue, as previously discussed, I feel that the application of trypsin will develop in usage and scope and soon become a more common adjunct to routine treatment in both the dental and the medical professions.

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AMELOBLASTOMA OF THE MANDIBLE

Report of a Case

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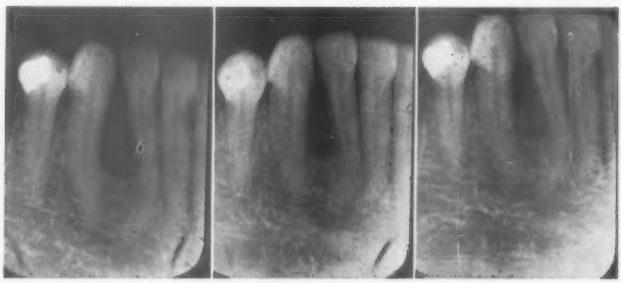
AMELOBLASTOMAS of the mandible have been described in the literature by Small,[1] Schweitzer,[2] Salman and Salman,[5] Bernier and Tiecke,[6] Lucas,[8] and many others. In most of the cases reported the tumors had become evident because of subjective symptoms, such as pain or swelling after the lesions had reached quite large proportions. The case presented here is of interest because the tumor was discovered early by routine x-ray examination while it was still quite small.

As pointed out by Small and associates,[9] it is important that every cystic-appearing lesion be examined microscopically. The case presented here further emphasizes this point.

CASE REPORT

Routine dental roentgenograms of a 39-year-old white woman were taken on March 2, 1958. These showed a sharply outlined roentgenolucent area between the left lateral incisor and the canine (Fig. 1, *A*). This looked very much like a parodontal cyst. Further examination of the patient's mouth revealed a slight, nontender, firm swelling over this area. The gingiva was of normal color.



A. *B.* *C.*

Fig. 1.—*A*, Intraoral roentgenogram of area before operation. *B* and *C*, Intraoral roentgenograms taken three months and six months after operation.

The patient was referred to this office on April 4, 1958, for removal of what was believed to be a "cyst." Under local anesthesia, a gingival flap was turned down, extending from the left central incisor to the first premolar, and a thick, membranous, cystic sac was

xposed in a bony crypt between the lateral incisor and the canine. This was enucleated in one piece. The sac was gray in color and very thick; it contained some dark brown fluid. The specimen was placed in 10 per cent formalin and sent to the laboratory for analysis.

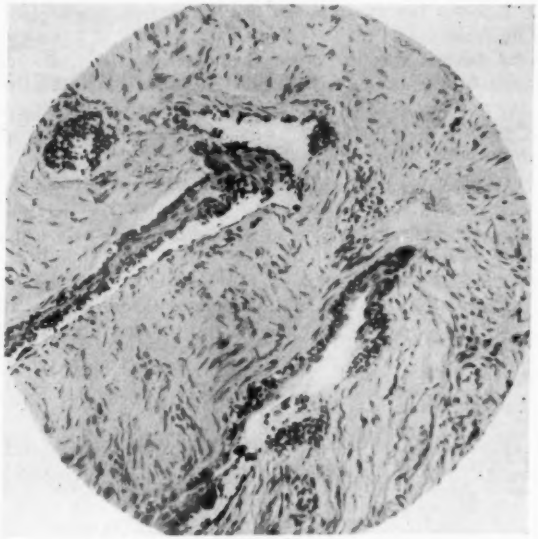


Fig. 2.

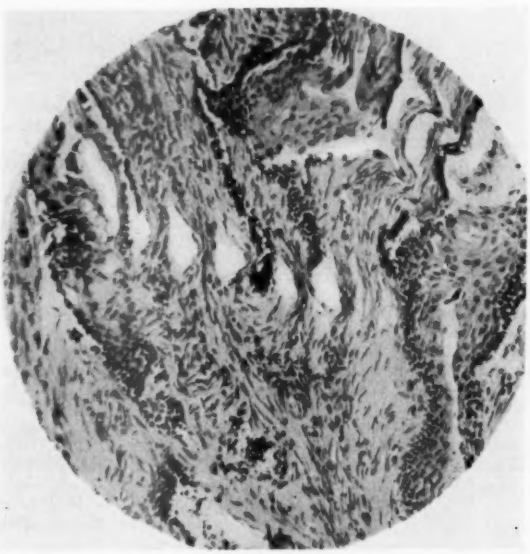


Fig. 3.

Figs. 2 and 3.—Two sections of ameloblastoma showing typical epithelial structures separated by dense connective tissue. (Magnification, ×100; reduced ¼.)

The resulting cavity was clean, smooth, and free of further debris or soft tissue of any kind. A square piece of Gelfoam was inserted, the soft tissues were approximated, and the wound was closed with continuous 0000 black silk sutures. Healing was uneventful. When the sutures were removed after five days, the incision was observed to be healing well.

Pathologist's Report.—The specimen consisted of a translucent piece of white tissue measuring 6 by 5 by 3 mm.

Microscopic examination revealed rather dense fibrous tissue, chromatically inflamed which contained numerous large and small epithelial islets with or without central keratinization (Fig. 2). In occasional areas, in the larger epithelial islands, central spongiosis with formation of stellate cells was seen (Fig. 3). The peripherally placed cells were cuboidal and hyperchromatic.

Impression: Ameloblastoma.

The patient was recalled on April 24, 1958. I explained very carefully to her and her husband that the "cyst" was of a type that sometimes recurs and told them of the importance of follow-up x-ray examinations.

Postoperative films were taken on June 14 (Fig. 1, *B*) and again on September 9 (Fig. 1, *C*). It was noted that the area of translucency was apparently filling in with new healthy-appearing bone. The patient is to have x-ray examinations periodically for three years.

SUMMARY AND CONCLUSIONS

A case of ameloblastoma of the mandible in a 39-year-old white woman has been presented. The roentgenogram of the lesion revealed a typical parodontal cyst, rather innocuous in appearance. Microscopic examination showed this to be an ameloblastoma of a very fibrous nature. Further surgery was not performed at that time, but the patient is to be followed by x-ray check-up. Bernier[10] has described a similar case in which there was a recurrence after seven years.

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HODGKIN'S DISEASE TREATED WITH NITROGEN MUSTARD AND RADIATION, COMPLICATED BY OSTEOMYELITIS OF THE MANDIBLE

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CASE REPORT

PATIENT S. V., a 33-year-old white man, was transferred to Detroit Receiving Hospital from Henry Ford Hospital in Detroit, Michigan, on April 5, 1955. The patient stated that he had experienced pain, tenderness, and swelling of the right mandible for the past three months.

Past History.—The patient stated that he had been well until 1949, when he noted a painless enlargement of the right side of his neck. He went to his family physician, who biopsied the node. The diagnosis was Hodgkin's disease. The patient received six x-ray treatments at St. Mary's Hospital in Detroit, Michigan. He felt better until six months later, when he noted weakness, weight loss, coughing, fever, night sweats, and nervousness. His weight in 1949 was 162 pounds; by April 5, 1955, it was 128 pounds. The symptoms had remained until this hospital admission, with only short remissions after courses of therapy; the fever never remitted. The patient saw several physicians but received no specific therapy until he went to Henry Ford Hospital in 1951. He had been treated there until April, 1955, when he had been transferred to Detroit Receiving Hospital because he was financially unable to continue therapy at Ford Hospital. At Ford Hospital he received several courses of x-ray therapy to the mandible, neck, chest, back, abdomen, and inguinal regions, the last in December, 1954. He also received a number of courses of nitrogen mustard therapy, the last of which was given in the fall of 1953. At that time he developed a number of blisters over his body, and therapy was discontinued. Late in 1954 he was given 20 mg. of TEM in divided doses. The patient was placed on cortisone, 25 mg. three times a day, for three weeks in August, 1954, and from January, 1955, until he was admitted to Detroit Receiving Hospital. He had received several transfusions for maintenance and periodic courses of antibiotics. The patient stated that after each course of therapy he felt better for a few months but then became worse again.

In December, 1954, he noted loosening of the teeth in the right mandible, and shortly afterward two of his teeth fell out and an ulcer was noted in the

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right mandible, in conjunction with swelling and pain. He subsequently developed a severe ulcerative osteomyelitis of the right mandible. The patient also complained of occasional dull aching back pain which radiated into the left leg through the bone to the knee for the past year and into both legs for the past three weeks. Also, he had occasional sharp but short-lived pain in the left side of his chest of several months' duration; this pain was not related to respiration or food intake.

Physical Examination.—Significant physical findings included a chronically ill appearance, slight pallor, obvious weight loss, no purpura, and facial asymmetry. There was a 1 by 3 cm. defect, with a depth of approximately 1.5 cm., on the superior border of the right mandible in the edentulous premolar region which appeared necrotic. The buccal, lingual, and facial areas in this region were tender to palpation, with diffuse induration of the right submandibular region. Bilateral submandibular nodes measuring up to 2 cm. were palpable, and the cervical nodes were shotty. Several nodes measuring 1 to 2 cm. were demonstrated in the left axilla. The liver was down 8 cm. and the spleen was down 7 cm. on inspiration, and both were tender and firm. The lower abdominal wall was slightly edematous. A mass-like loop of bowel was palpated just to the right of the midline in the middle of the abdomen. The patient was hypersensitive in both CVA areas. There was minimal sacral edema.

Roentgenographic Examination.—On admission, the impression of the visualization of the chest was recorded as "no definite evidence of active parenchymal disease or hilar adenopathy." Also, the impression gained from routine studies of the mandible was that there was a destructive process involving the right body of the mandible and that differential diagnosis was between a neoplastic and an infectious process. A gastrointestinal series done on May 3, 1955, indicated an "enlarged spleen and liver, with essentially negative upper G.I. series." The lumbar spine and sacroiliac articulations appeared normal. On re-examination on May 31, 1955, the chest had changed markedly. The right leaf of the diaphragm was not seen, and there was a pleural effusion on the right side with perhaps some loculation. The upper border of the effusion had a somewhat unusual appearance, the nature of which was not clearly understood, and the fluid appeared to be fairly well posteriorly. It was recommended the patient be tapped and re-examined. Thoracentesis was performed in the ninth intercostal space at the posterior axillary line on the right side on June 8, 1955, and 2,000 c.c. of amber-colored fluid was drawn off. The roentgenographic findings at this time were that there was much less pleural effusion on the right side and that there appeared to be some parenchymal density well to the posterior. Thoracentesis was performed again on June 28, 1955, and 2,500 c.c. of serosanguineous, watery fluid was removed. The posteroanterior and lateral chest views at this time indicated a reaccumulation of fluid in the right hemithorax.

Laboratory Examination.—A total of 127 specimens were taken for laboratory analysis. Twenty-three of these were for transfusions administered during

the four months the patient remained in the hospital until he died on Aug. 16, 1955. Summation and averages of laboratory findings are as follows:

	High	*Low*	*Average*
Hemoglobin	11.5	6.3	8.1
Red blood cells	3.98	2.62	3.40
White blood cells	7,200	1,750	3,200
Neutrophils	89	77	85
Lymphocytes	15	3	12
Monocytes	19	4	7
Reticulocytes	3.4	1.8	2

The neutrophils showed a toxic reaction throughout
Negative serology
Clotting time—8½ minutes
Clot retraction—15 minutes, completed 2 hours
Platelets—292,000
Sedimentation rate 15 minutes, 37 mm. 60 minutes, 129 mm.
Dextrose 78 mg.
Urea nitrogen 9 mg.
Alkaline phosphatase 7.4-13.4 Bodansky units
Bilirubin 1.8 mg.
Bromsulphalein 16 per cent of dye retained in 45 minutes
Serum protein 5.5 Gm.
Serum albumin 3.9 Gm.
Serum globulin 1.6 Gm.
Fungus growth from mandible—No growth
Anaerobic culture from mandible—No growth
Aerobic culture of mandible—Alpha streptococcus, diphtheroids, *Bacillus proteus*
Plasma iron 50 gamma per cent
Urine
    4 to 6 white blood cells
    1 to 2 squamous epithelium
Feces occult blood
    Confirmative ++++
    Presumptive +

Treatment and Course.—The patient was admitted to Detroit Receiving Hospital on April 5, 1955. He was placed on the medical service and carried by the hematology department. Blood and roentgenographic studies were ordered. The patient was maintained on antibiotics, vitamins, analgesics, cortisone, ACTH, low-salt diet, Thorazine, transfusions, and additional supportive therapy during his hospitalization. The oral surgery management consisted of palliative therapy during the patient's stay in the hospital. The oral surgery note made on admission stated that the denuded bony defect of the body of the right mandible was suppurating and it was believed that the defect was the result of osteoradionecrosis (Fig. 1). The area was irrigated with normal sterile saline and lightly packed with iodoform gauze which was lubricated with 1 per cent Neo-Cortef ointment. It was deemed inadvisable at this time to perform a sequestrectomy or approximation of tissue through suturing. Rather, it was decided to keep the drain in the wound to keep food from entering and to treat the bony crypt palliatively. This type of conservative treatment for the mandible was continued throughout the patient's hospitalization. At times the iodoform gauze was alternately moistened with eugenol, Benzocaine, Furacin, Varidase, Cyclaine, or Neo-Cortef to relieve pain. On June 10, 1955, the right inferior alveolar and buccinator nerves were blocked with 1.5 c.c. of 95 per cent ethyl alcohol. The possibility of a slough from this procedure due to the previous deep therapy was recognized, but it was deemed

advisable to risk it, since it was of secondary importance with the patient in the terminal stage of his illness. Alleviation of pain was of far greater importance. The patient received a limited amount of relief from this procedure.

Fig. 1.

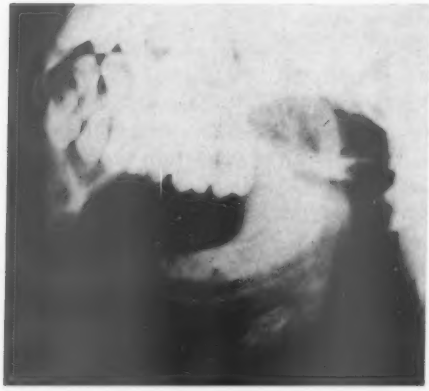

Fig. 2.

A repeat injection was made two weeks later, with a better result. A biopsy specimen of a section of bone which had sequestrated from the right mandible on April 28, 1955, was diagnosed as "necrotic bone." On June 3, 1955, the lower left central incisor was removed without anesthesia. The lower right central incisor was removed on July 21, 1955, since the patient had sustained a pathologic fracture of the mandible the previous night while sleeping (Figs. 2, 3, and 4). Because of the previous alcohol injection, there was no pain attached to this fracture. During the patient's last two months he found it very difficult to retain food, even though he was given a liquid diet. During his

Fig. 3.

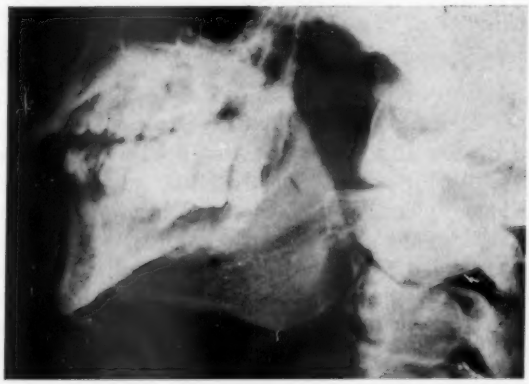

Fig. 4.

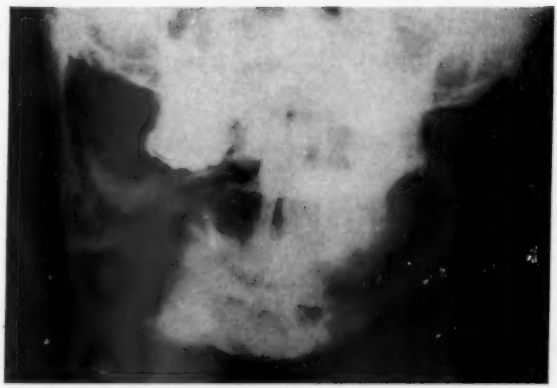

Fig. 5.

stay in the hospital there was a continuous decline in his general condition (Fig. 5). He died at 3:50 A.M. on Aug. 16, 1955, after a four-month confinement in Detroit Receiving Hospital.

Postmortem Observations.—The following is a combined anatomic and histologic diagnosis received from the pathologist:

1. Hodgkin's disease involving spleen, lungs, bone marrow, and lymph nodes
   (a) Hydrothorax, bilateral
      (1) Atelectasis of lungs
   (b) Fibrosis, postirradiation, in lungs, lymph nodes, and spleen
   (c) Ulcer, decubitus, in right scapular area
   (d) Hypoplasia of bone marrow
   (e) Anemia
   (f) Cachexia
2. Osteomyelitis of right mandible
3. Hemosiderosis, posttransfusion, of liver, spleen, and lungs.

RESULT

The clinical picture of a chronic wasting disease with weight lose, anemia, and nutritional disturbances was confirmed at autopsy by the findings of changes in the lungs, spleen, lymph nodes, and bone marrow which were diagnostic of Hodgkin's disease. The fibrosis seen in the spleen, lungs, and lymph nodes reflects in part the x-ray and nitrogen mustard treatment that this patient had received. Hypoplasia of the bone marrow at least partially explains the extreme anemia which during the course of this illness had necessitated multiple blood transfusions. As evidence of multiple blood transfusions, there was hemosiderosis of the liver, spleen, and lungs. We may justify the diagnosis of osteomyelitis of the mandible by citing the osteoradionecrosis as a predisposing factor. This case was managed throughout by the teamwork of persons from several departments of both the medical and the surgery services who cooperated to the fullest extent for the betterment of the patient's condition.

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PREGNANCY TUMOR SUPERIMPOSED UPON A NEVUS

Report of a Case

Newton E. Allen, D.D.S., F.A.C.D., Selma, Ala.

PREGNANCY tumors are thought to be caused by hormonal stimulation during gestation,[1-7] and they occur rather frequently in women with gingivitis of pregnancy. They occur on the skin and mucous membrane of the nose, tongue, lips, gingiva, and rectum.[7] The oral lesion, which is reddish blue in color, tends to decrease in size postpartum and to recur in another pregnancy. These lesions resemble granuloma pyogenicum, both clinically and microscopically. According to Kerr,[8] the tumor of pregnancy cannot be distinguished from the granuloma pyogenicum by histologic findings. Fabe[7] thinks that these tumors are primarily hemangiomas, usually of the cavernous type, and cannot be histologically differentiated from them. Blum[1] refers to these lesions as angiogranulomas.

Ziskin and Nesse,[5] Fabe,[7] and Thoma[9] have reported pregnancy tumors associated with nevi and have supplied evidence that nevi are predisposed to tumor formation during gestation.

The interesting case reported here involved a large pregnancy tumor superimposed upon a nevus. The methods employed for its excision, control of anticipated excessive bleeding, and management of the surgical defect caused by its removal are presented.

CASE REPORT

A 22-year-old Negro woman in the fifth month of her second pregnancy was seen on Aug. 17, 1954, having been referred by her physician for diagnosis and treatment of a tumor in the right maxillary area.

History.—The patient first noticed the mass two years earlier, during the third or fourth month of her first pregnancy. After parturition, the tumor decreased in size but did not disappear. It became greatly enlarged during the current pregnancy, beginning about the second month. A large nevus on the right side of the face had existed since birth. The patient stated that she had never been ill in her life, and the family history was noncontributory.

Examination.—The right side of the face was obviously enlarged. A nevus flammeus covered the external as well as the internal surfaces of the upper lip (Fig. 1) and spread over the cheek from the level of the occlusal plane of the teeth to the lower eyelid. Externally, it was reddish brown and slightly elevated. Almost its entire border was outlined by a definite line of demarcation. Other than the color and the slight elevation, the gross

appearance of the surface was that of normal skin. Palpation, however, revealed it to be puffy or spongy. On the inner surface of the cheek the nevus was reddish and granular. It also involved the mucobuccal fold and the labial gingival tissue to the median line.

Fig. 1.—Nevus flammeus involving right upper lip and cheek (Aug. 17, 1954).

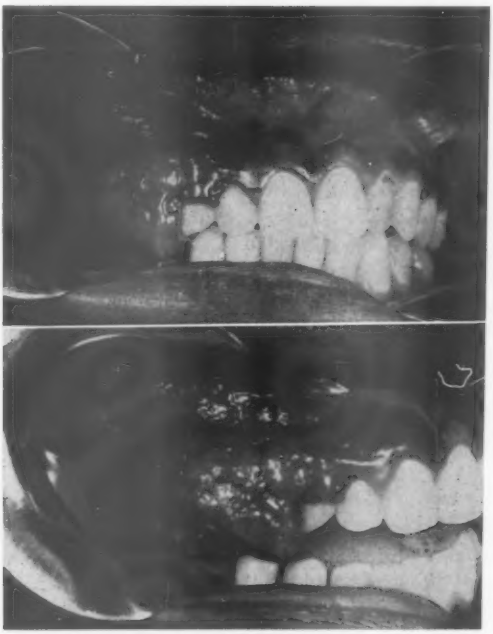

Fig. 2.—Photographs of pregnancy tumor superimposed upon nevus (Aug. 17, 1954).

A large mass extended from the upper right lateral incisor to the tuberosity of the maxilla and covered the occlusal surfaces of several teeth (Fig. 2). The tumor appeared primarily on the buccal side of the arch but involved the palatal tissues to a lesser degree. It interfered with mastication, it bled easily, and on palpation it could be felt to pulsate.

The anterior portion of the tumor was bright pink, firm, and lobular. It was apparently covered by rather smooth and shiny epithelium, whereas the posterior part was similar in color and texture to the mucosal covering of the oral surface of the nevus.

There was an area of hypertrophied tissue in the lower right molar region. Several teeth were affected by advanced caries, and calculus formation was slight.

Roentgenographic examination (Fig. 3) revealed spacing of the upper right cuspid and first premolar. Some resorption of bone between these teeth was evident.

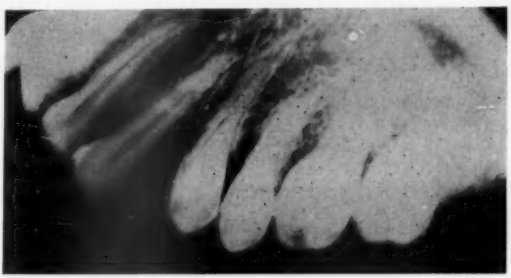

Fig. 3.—Roentgenogram showing spacing of cuspid and first premolar (Aug. 17, 1954).

Except for the orofacial lesions, the patient, whose pregnancy was progressing normally, was in excellent health. The referring physician reported that the findings of a physical examination, including laboratory studies, were within normal limits.

Biopsy.—On the date of the initial examination, tissue for microscopic study was removed under local anesthesia. An attempt was made to obtain a specimen that would comprise a representative section of the tumor mass and the surrounding mucoperiosteum. The tissue was placed in bottle No. 1 containing 10 per cent formalin solution. Profuse bleeding was encountered, and it was necessary to suture a gelatin sponge (Gelfoam) dressing into place to control the hemorrhage. A section of the hypertrophied tissue in the right mandibular molar area was also removed for examination and placed in bottle No. 2. The material was submitted to Colonel Joseph L. Bernier at the Armed Forces Institute of Pathology, and his report, dated Aug. 21, 1954, follows:

> *Microscopic:* Two sections of tissue are examined. The section from the tumor mass (1) is characterized by numerous vascular channels of varying sizes. There is some endothelial proliferation with a few scattered chronic inflammatory cells. The tissue is markedly edematous and is covered by stratified squamous epithelium exhibiting acanthosis and parakeratosis. The other section (2) is covered by hyperplastic stratified squamous epithelium and in one area shows surface ulceration. The underlying connective tissue is heavily infiltrated by lymphocytes and plasma cells. The recognizable fibrous tissue shows foci of myxomatous degeneration.
>
> *Diagnosis:* (1) Pregnancy tumor; (2) hyperplastic oral mucosa.

Treatment and Course.—On Aug. 23, 1954, the sutures placed at the time of biopsy were removed and excision of the tumor was scheduled for Aug. 25, 1954. The patient failed to come on that day and was not seen again until Sept. 20, 1954, when she came to make another appointment. On the afternoon of Sept. 22, 1954, she was given a prophylactic intramuscular injection of 5 mg. of Adrenosem Salicylate. On the following day premedication consisted of 0.1 gram (1.5 grains) of pentobarbital sodium, administered

orally, and an intramuscular injection of 5 mg. of Adrenosem Salicylate one hour before the operation.

Local anesthesia was obtained by blocking the posterior superior alveolar, anterior superior alveolar, anterior palatine, and nasopalatine nerves and by infiltrating the tissues surrounding the tumor with 2 per cent lidocaine hydrochloride with epinephrine 1:50,000.

Fig. 4.—Gross appearance of specimen after removal (Sept. 23, 1954).

Fig. 5.

Fig. 6.

Fig. 5.—Operative field immediately after excision of tumor, extraction of two teeth, and reduction of alveolar bone between the cuspid and first premolar.

Fig. 6.—After gelatin sponge had been placed over the crest of the ridge, loose sutures were attached to the previously undermined mucosa and to the palatal soft tissues (Sept. 23, 1954).

Fig. 7.—Surgical cement packed over the sutures and gelatin sponge protects the defect created by removal of the tumor (Sept. 23, 1954).

An incision started in the mucoperiosteum covering the outer surface of the maxilla in the tuberosity region was carried down to the bone and extended horizontally in an anterior direction to a point near the mesial surface of the first molar. It was then curved upward, high into the sulcus, to circumvent the lobular portion of the tumor and downward again to the gingival border at the median line. The palatal incision was made high up, beginning at the posterior end of the buccal incision distal to the second molar and extending anteriorly to the central incisor. The entire soft tissue mass between the incisions, including the residual soft tissue from the interproximal spaces (Fig. 4), was removed with a contra-angle, double-end saw type of curette. The alveolar crest was then reduced by curettage. Since soft tissue still existed between the cuspid and the first premolar, both of which were loose, these teeth were extracted and the interproximal tissues were removed with rongeur forceps until healthy bone was reached (Fig. 5).

In order to cover the exposed bone between the lateral incisor and the second premolar without lowering the muscular attachments excessively, the buccal mucosa was undermined to permit its being brought closer to the crest of the ridge. A piece of dry gelatin sponge (Gelfoam) was cut to fit the crest of the ridge, and the assistant held it in position with dry instruments while silk sutures were attached to the undermined mucosa and the palatal soft tissue. The sutures were passed over the gelatin sponge and tied loosely to avoid tension (Fig. 6). Surgical cement, to which had been added tannic acid powder, was mixed to a thick consistency and applied under pressure around the teeth and over the surrounding soft tissues (Fig. 7). During the entire procedure, bleeding was moderate.

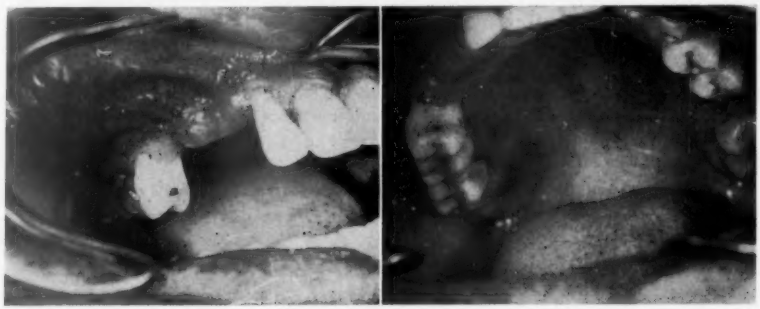

Fig. 8.—Two views of area after healing. Healthy tissue covers the alveolar ridge, and the depth of the labioalveolar sulcus is adequate.

The patient was seen on the following day. She had experienced very little pain, no bleeding, and only minimal swelling. She visited the office on Sept. 27, Sept. 30, and Oct. 3, 1954. No complications were encountered. On Oct. 7, 1954, the surgical dressing and sutures were removed and a new dressing was applied. On October 14 the dressing was eliminated.

Results.—Healing was satisfactory when the patient came to the office on Oct. 28, 1954, and healthy tissue covered the defect that had been created by removal of the tumor and extraction of the teeth (Fig. 8). She was advised to return for periodic observation and to contact her dentist for routine dental care.

A questionnaire was sent to the patient, and a reply dated Jan. 15, 1958, stated that there had been no recurrence of the lesion, even though she now had a third child.

COMMENT

In the case just reported, a large pregnancy tumor superimposed upon a nevus was vascular to the extent that palpation revealed it to pulsate.

Anticipated excessive bleeding was apparently controlled by the prophylactic administration of Adrenosem Salicylate, the use of a local anesthetic solution containing epinephrine, and the application of a surgical cement to which tannic acid powder had been added.

To protect the surgical defect without seriously reducing the depth of the buccoalveolar sulcus, a piece of gelatin sponge was placed over the crest of the alveolar ridge and held by passing silk sutures from the buccal mucosa to the palatal soft tissues and by placing surgical cement over the entire area. Apparently, the gelatin sponge acted as a matrix for the absorption of blood, which resulted in bridging of the ridge with granulation tissue followed by epithelization. The possibility exists, however, that the gelatin sponge merely provided a space under the surgical pack to permit healing by granulation.

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410 CHURCH ST.

RECONSTRUCTION OF MANDIBLE FOLLOWING BONE LOSS DUE TO OSTEOMYELITIS IN THE LINE OF FRACTURE

Harold F. Bosco, D.M.D., New Britain, Conn.*

THE oral surgeon is often called upon to reconstruct jaw deformities which result from trauma, neoplasms, and infections. The mandibular defects are more interesting to the oral surgeon, since in most cases a surgical approach may be used to correct them, in contrast to maxillary deformities that can be readily obturated with prosthetic appliances.

In the case to be presented the mandible was reconstructed by means of a three-step surgical procedure combined into one operation.

CASE REPORT

Patient P. B., a 46-year-old white woman, was first seen in my office on Nov. 26, 1957. She appeared distressed and emotionally disturbed as the result of a jaw fracture which she had received in July, 1957. The patient's chief complaints were inability to wear dentures, inability to masticate food properly, and especially the disfigurement caused by a change in the contour of the lower third of her face.

Past History.—The patient had sustained a fracture of the lower right side of the mandible in the region of the mental foramen in July, 1957. She reported to a hospital in a neighboring community, where treatment of the fractured mandible was instituted by an orthopedic surgeon. The treatment consisted of applying a stainless steel bone plate attached with four screws, two on each side of the fracture site. Results of this procedure were unsatisfactory, since the bone plate had to be removed due to the development of osteomyelitis at the site of the fracture. The patient developed extraoral and intraoral fistulas, which the attending physician treated with large amounts of antibiotics. Several sequestrectomies and saucerizations resulted in a considerable amount of bone loss and disfigurement of the patient's face (Fig. 1).

Oral Examination.—Nonunion of the mandible, with a considerable amount of scar formation in the submaxillary region on the right side was noted. The patient complained of numbness of the lower lip and stated that she had seen no drainage, either intraorally or extraorally, for a period of two weeks. Bone grafting was decided upon, and hospitalization was advised.

Hospital Course.—On Jan. 13, 1958, the patient was admitted to the New Britain General Hospital for treatment of her jaw deformity.

Physical examination on admission revealed a middle-aged white woman in no acute distress, with disfigurement involving the lower third of her face. The chest was clear to

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percussion and auscultation. The heart had normal sinus rhythm, and no murmurs were present. There was no history of systemic disease.

Laboratory studies revealed a red blood count of 4.2 million, hemoglobin 81 per cent, and a white blood count of 12,550; leukocytes showed 86 per cent neutrophils, 1 eosinophil, 10 lymphocytes, and 3 monocytes. The nonprotein nitrogen was 30 mg. per cent. The patient had group A blood.

Fig. 1.—Preoperative photograph showing facial deformity and shift of mandible to the right side.

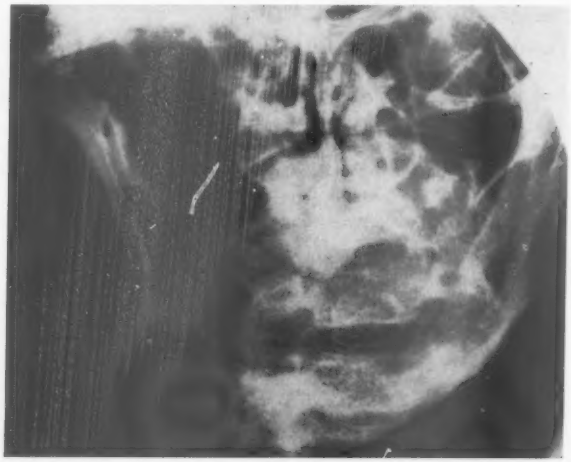

Fig. 2.—Anteroposterior view showing extensive bone loss of mandible on right side.

Preoperative roentgenograms revealed considerable loss of bone structure in the lower right jaw; also, an isolated bone fragment was found to be present (Fig. 2). X-ray examination indicated that the chest was normal, and the heart was reported to be of normal size.

Operation.—On Jan. 14, 1958, following the usual preoperative medication, general anesthesia was induced and continued by means of a nasoendotracheal tube. A three-step operation was performed. In the first stage circumferential wires were placed around the solid portion of the mandible on the left side, and wires were inserted through bur holes drilled in the pyriform nasal crest of the maxilla on the right and left sides. The wires were allowed to remain long, and after the upper and lower dentures were inserted

Fig. 3. Fig. 4.

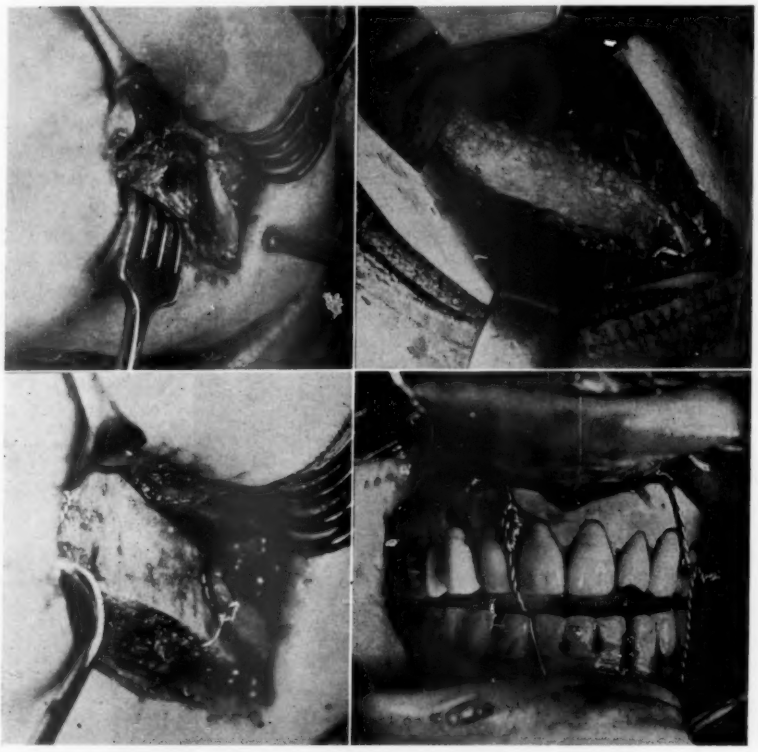

Fig. 5. Fig. 6.

Fig. 3.—Site prepared for receiving bone graft.
Fig. 4.—Anterior iliac crest from which bone graft was obtained.
Fig. 5.—Graft in place, stabilized with wire ligatures.
Fig. 6.—Dentures in position and immobilization of the mandible.

(with some difficulty) the maxillary and mandibular wires were brought together. The second stage, in which a completely new setup was used, consisted of preparing the site to receive a bone graft. This was done by excising the scar in the skin in the submaxillary region, locating and preparing the ends of the posterior and anterior bone fragments, and removing an isolated bone splinter and scar tissue between the bony ends (Fig. 3). In the third stage the bone graft from the anterior crest of the ilium was mobilized by an orthopedic surgeon (Fig. 4). The graft was then inserted into the prepared area of the mandible, where it was stabilized by means of wire ligatures (Fig. 5). The wound was closed in

Fig. 7.—Postoperative roentgenogram showing immobilization of mandible and bone graft in position.

Fig. 8.—Postoperative photograph showing final improvement of patient's appearance.

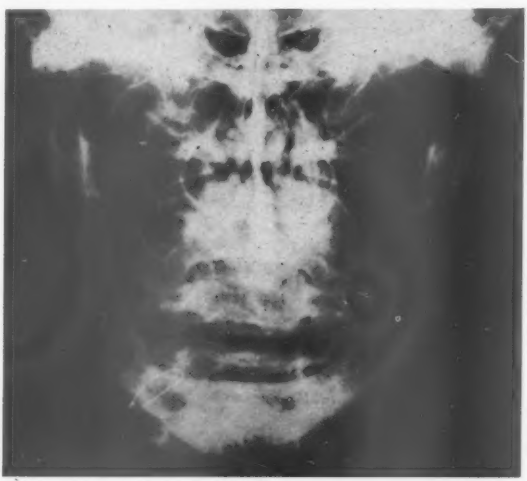

Fig. 9.—Anteroposterior view taken on Dec. 9, 1958, showing bone grafts to be in good position and vital.

layers, and pressure dressings were applied. The intraoral wires were secured to immobilize the mandible and to bring the upper and lower dentures into proper occlusal relationship (Fig. 6).

Postoperative Course.—The patient was given general supportive care. Antibiotic therapy was prescribed and continued for a period of twelve days. During this time the patient was comfortable, in spite of the fact that she was unable to eat in the usual manner. Postoperative roentgenograms were taken on Jan. 20, 1958. These showed the bone graft to be in good position and adequately stabilized (Fig. 7). The patient was discharged on Jan. 25, 1958, with the intraoral appliances still in place. On March 12, 1958, all wires were removed. The patient's appearance was greatly improved (Fig. 8). She was now able to chew, wear dentures, and once again carry on a normal life. Most recent roentgenograms, taken on Dec. 9, 1958, showed the bone graft to be vital (Fig. 9).

DISCUSSION

In reconstructive surgery of the mandible the basic principles set forth by Thoma[1] in his textbook on oral surgery must be adhered to in every detail when bone grafts are used. It is important, however, that surgeons involved in this type of surgery have additional knowledge relating to the occlusion of teeth and a deep understanding of the physiology of the masticatory system. It is for this reason that the oral surgeon should be the team captain in reconstructive surgery involving jaw deformities.

My gratitude goes to the Orthopedic Department of New Britain General Hospital and especially to Dr. Maxwell E. Hagedorn, orthopedic surgeon, for mobilizing the bone graft and for his assistance in this case. Photographs were taken by Mr. Alfred Spitzer, Clinical Photography Department of New Britain General Hospital.

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300 Main St.

EXODONTIA

OSTEOSCLEROSIS IN EDENTULOUS MANDIBLE SIMULATING CARIOUS UNERUPTED CUSPID

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THE case report that follows is presented to illustrate an error in diagnosis and the ill-conceived treatment to which it led. Initial clinical and roentgenographic examinations prompted a decision in favor of surgical intervention for removal of an unerupted tooth which, in reality, did not exist.

CASE REPORT

Patient S. K., a 59-year-old woman, was referred by her dentist on Dec. 15, 1949. She had an incised bleeding wound in the region of the mandibular left cuspid and first premolar and was in great pain.

History.—The patient suffered pain whenever her complete lower denture was subjected to masticatory stress. This pain was experienced posteriorly and superiorly on the left side of the face. The patient's remaining six anterior teeth had been removed three years previously, in 1946. When the patient ultimately detected a hard substance, she visited her dentist again and accused him of permitting a portion of a root to remain in her jaw. After due clinical and roentgenographic examinations, the dentist proceeded to operate to remove this hard substance. After considerable manipulation and some apparent trauma, the patient was referred to us for removal of the "root."

The patient's family history was noncontributory.

Examination.—The first intraoral film failed to reveal any roentgenographic delineation of a retained root. The alveolar border was irregular and appeared to be quite dense. A second film, taken anteriorly in a longitudinal direction, disclosed the presence of what appeared to be a carious unerupted cuspid in a mesiodistal position with the apex close to the mandibular border (Figs. 1 and 2). An extraoral plate confirmed the findings of the intraoral film (Fig. 3). Surgical removal was advised.

General Physical Findings.—The patient had recently been examined by her physician, who requested that a local anesthetic be used for any surgery that might be necessary.

Treatment and Course.—The area was anesthetized with 6 c.c. of 2 per cent procaine hydrochloride with Adrenaline, which was distributed at the left mandibular and left mental foramina, the anterior labial and lingual mucosal areas 1 cm. beneath the ridge, and also through the skin at the inferior mandibular border. After an adequate mucoperiosteal flap had been retracted, the "unerupted tooth" was outlined with sharp hand chisels. The tissue

encountered was extremely dense, and on several occasions a tendency to slip was noted. Mallet blows were then directed to the area, and finally surgical drills and burrs were employed. At this point, when no tissue resembling enamel came into view, a magnifying glass was used to determine whether or not any histologic form of tooth structure was present and

Fig. 1.

Fig. 2.

Figs. 1 and 2.—Intraoral views of left side of mandible. The longitudinal view shows what appears to be an unerupted tooth with an absorbed or mutilated crown. An attempt to remove a "root" had been made by the referring dentist.

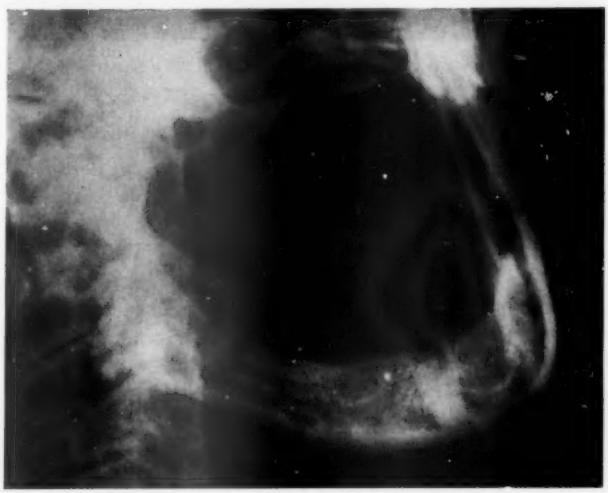

Fig. 3.—Left view of mandible showing the outline of an "unerupted tooth" with an absorbed or mutilated crown. The apex appears to extend to the inferior border of the mandible.

to rule out the possibility that there might be an anomalous maldevelopment of embryonic tooth remnant establishing fusion with osseous structure. No such junction or line of union could be identified. It was at once apparent that we were dealing with a bone condition which

resembled an impacted tooth and that failure to take a positive stand with respect to diagnosis would terminate in useless tissue destruction with possible nerve injury and mandibular fracture. Bearing in mind the original complaint, we reduced the entire bone area with surgical burrs and made it thoroughly round for a distance of about 2 mm. beneath the adjacent ridge. Hot saline sponges were applied, and the tissue flaps were sutured with silk.

The patient experienced a rather severe swelling and pain postoperatively, but these symptoms gradually diminished during the next ten days. Codeine, ¼ grain to be taken every three hours, was prescribed. The sutures were removed on the fifth day, and on a subsequent office visit the patient was wearing her denture comfortably. Two years later the patient was still free of symptoms (Fig. 4).

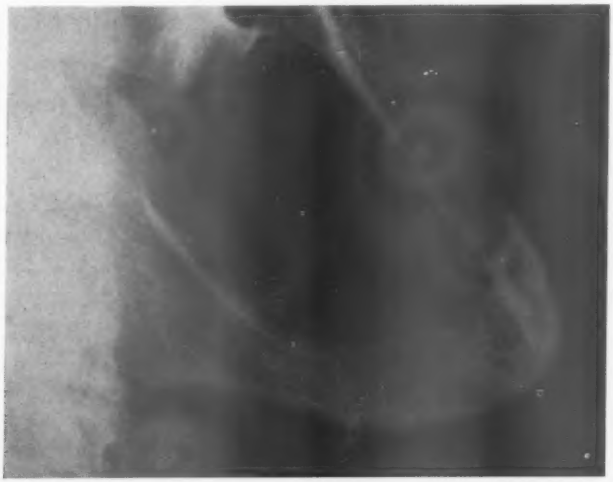

Fig. 4.—View of left mandible two years following surgery. The patient at this time was free of symptoms.

CONCLUSIONS

The case just presented demonstrates the fallacy of permitting operative tactics to proceed along predetermined lines based solely upon clinical and roentgenographic impressions that may be erroneous. This case illustrates the need for resourcefulness on the part of the operator. He should reanalyze the diagnosis during the operation, and, if necessary, the stubborn execution of the original plan, which might result in unwarranted and needless tissue destruction with possible nerve injury, mandibular fracture, or other deleterious effects of serious consequence to the patient, should be abandoned in favor of a more realistic procedure.

189 HALSEY ST.

ORAL MEDICINE

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BLEEDING DUE TO DEFICIENCY OF PLASMA THROMBOPLASTIN ANTECEDENT (PTA) AND PLASMA THROMBOPLASTIN COMPONENT (PTC)

Report of Nine Cases

Ronald F. Steg, D.D.S., Robert J. Gores, D.D.S., John H. Thompson, Jr., Ph.D., and Charles A. Owen, Jr., M.D., Rochester, Minn.

ABNORMAL postoperative hemorrhage occurring after oral operations usually results in a distressful situation for the surgeon, the patient, and the patient's family. Fortunately, most postoperative bleeding can be arrested by such simple methods as gauze packing, pressure, suturing, or the use of various hemostatic agents.

Occasionally, however, underlying pathologic defects in the blood-clotting mechanism exist, and common methods of hemostasis are fruitless. Unfortunately, at present no simple laboratory test is available for detection of these defects; therefore, an accurate diagnosis is not easily established. The diagnostician must rely on a careful history, for this often is the only clue that will suggest the actual pathologic changes.

It is generally agreed that the clotting of blood begins by means of an interaction between platelets and a series of plasma proteins, namely, antihemophilic globulin (AHG), plasma thromboplastin component (PTC), plasma thromboplastin antecedent (PTA), and possibly others. Thromboplastin is the product of this first step in clotting. In the second, or cothromboplastic step, the newly formed plasma thromboplastin combines with a labile protein, two stable proteins, and calcium. The product of this step permits conversion of prothrombin to thrombin, and finally the thrombin activates the conversion of fibrinogen to fibrin clot.

Deficiency of antihemophilic globulin (AHG) leads to classic hemophilia, whereas lack of plasma thromboplastin component (PTC) gives rise to a

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hemophilia-like (hemophilioid) state that is clinically similar to classic hemophilia but distinct in its manifestations in vitro. The term "plasma thromboplastin component (PTC) deficiency" was used by Aggeler's group[1] to describe this new disease, while the term "Christmas disease" was proposed by Biggs and associates.[3] A deficiency in plasma thromboplastin antecedent (PTA), which was first described by Rosenthal and associates[6] in 1953, probably is caused by lack of a third plasma protein.

If any of these three plasma protein factors is missing, or if it is not present in sufficient quantity at the moment when it is needed, the generation of thromboplastin can be impaired. Therefore, as a direct result, the first step of the clotting mechanism, which is production of thromboplastin, does not take place at a sufficiently rapid rate to support the rest of the clotting "chain reaction."

In normal clotting, the available AHG is almost completely utilized during the clotting process, whereas PTA is utilized in limited amounts only, and the quantity of PTC remains relatively constant. Patients suffering from deficiencies of PTC and PTA usually respond well to the administration of fresh or stored plasma. Correction of a deficiency of AHG requires administration of fresh blood or plasma, for the AHG factor is extremely labile and its amount is reduced in stored blood or plasma. Deficiencies of AHG and of PTC are transmitted as sex-linked recessive traits by female carriers to males. As mentioned before, the clinical picture of the two syndromes is indistinguishable.[7] PTA deficiency is transmitted as an autosomal, dominant trait, with an apparently high degree of penetrance and variable expression. It is transmitted by either female or male to female or male progeny.[6]

While PTA differs in properties from both AHG and PTC, in general it bears a closer resemblance to PTC. PTA and PTC are not consumed during coagulation, they are present in serum, and they are fairly stable during storage. Treatment of either normal plasma or serum with barium sulfate or by Seitz filtration effects the complete removal of PTC, whereas only slight to moderate amounts of PTA are removed. AHG is not removed by these measures.[6]

Bleeding times, platelet counts, clot retraction, the concentration of calcium and fibrinogen, and the results of tourniquet tests and of one- and two-stage prothrombin tests on plasma are normal in AHG, PTC, and PTA deficiencies. The diagnosis of PTC and PTA deficiencies can be established best by the characteristic results obtained by the thromboplastin-generation test, although simpler procedures, including the "prothrombin-consumption test," often are useful.[5]

Since we became aware of our first case of postsurgical bleeding due to a deficiency of plasma thromboplastin antecedent (PTA) in 1955,[8] we have had occasion to study three more patients with this disease, in addition to five patients deficient in plasma thromboplastin component (PTC deficiency or Christmas disease).

Throughout this article "PTA deficiency" means a pronounced abnormality in thromboplastin generation in both serum and adsorbed plasma. To identify

one of these patients as definitely lacking the PTA factor, one would have to cross-check the patient's plasma with that of a patient identified as being deficient in PTA, as described by Rosenthal and associates. This has not been done in any of our cases.

PRESENT STUDY

A study was made of nine patients with deficiency of either PTA or PTC who had oral surgical procedures performed at the Mayo Clinic. Five of these patients had PTC deficiency (Christmas disease), and four had deficiencies of

TABLE I. CLINICAL DATA IN NINE CASES OF HEMOPHILIOID DISEASES

CASE	AGE (YEARS) AND SEX	TYPE OF DEFICIENCY	HISTORY OF HEMORRHAGE		OPERATION	PREOPERATIVE WHOLE BLOOD (ML.)	POSTOPERATIVE BLEEDING
			FAMILIAL	PERSONAL			
1	6 F	PTA*	0	+	Multiple extractions of deciduous teeth	250	0
2	21 F	PTC*	+	+	Extraction of remaining maxillary teeth and alveoloplasty	500	Mild 7 days postoperatively; stopped spontaneously
3	74 F	PTC*	0	+	Extraction of remaining maxillary teeth and alveoloplasty	500	Mild 6 and 16 days postoperatively; hematoma 19 days after surgery
4	17 F	PTC*	0	+	Four third-molar odontectomies	500	0
5	18 F	PTA	0	+	Root-canal filling and apicoectomy	0	Oozing 9 days postoperatively
6	29 M	PTC	0	+ ?	Extraction of remaining teeth and alveoloplasty	0	Moderate oozing 3 days postoperatively; hemorrhage 4 days postoperatively
7	31 M	PTA	+	+	One third-molar odontectomy	0	Oozing for 8 days; transfusion (500 ml.) 8 days postoperatively
8	47 M	PTC*	0	+	Multiple extractions	500	0
9	21 M	PTA	Family record not available	+	Four third-molar odontectomies	0	Moderate to severe oozing 7 days postoperatively; packed with hemostatic agent second postoperative day

*Diagnosed preoperatively.

the PTA type. Five of the patients were definitely known preoperatively to have a hemophilioid disease. Table I summarizes the essential data in these cases.

Only two of the nine patients had family histories of any bleeding tendency. However, eight of the nine presented definite personal histories of various episodes of bleeding after surgical procedures or trauma, and the information in the ninth case was suggestive but the patient was a poor historian and accurate information could not be obtained.

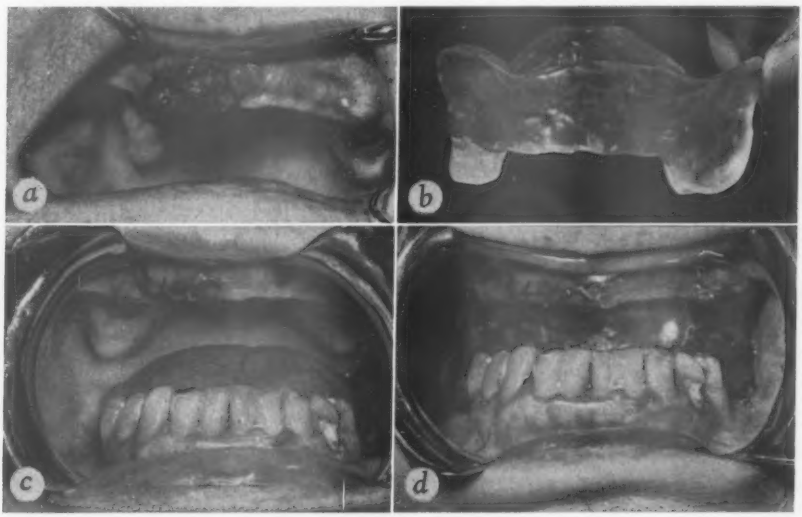

Fig. 1.—*a*, Hematoma of upper right alveolar ridge 2.5 cm. in diameter. *b*, Clear acrylic upper splint, with saddles extended into the lower posterior edentulous spaces. *c*, Hematoma removed and base of wound closed with 000 silk suture. *d*, Acrylic splint in place over sutured wound.

The five patients who had preoperative diagnoses of hemophilioid states received transfusions of whole blood prior to operation. Four of these patients had PTC deficiency (Christmas disease), and one was deficient in the PTA factor. Three of these five patients did not have any postoperative bleeding problems, one had slight hemorrhage seven days after operation which stopped spontaneously without additional treatment, and the remaining patient had slight continuous oozing from the operative site during the immediate postoperative period, with development of a hematoma (Fig. 1, *a*). This complication probably resulted from the fact that the remaining lower teeth traumatized the upper ridge during eating and while the patient was sleeping; therefore, an acrylic maxillary bite splint (Fig. 1, *b*) was fabricated for the patient, the hematoma was removed (Fig. 1, *c*), and the splint was put into place (Fig. 1, *d*). A transfusion also was given prior to removal of the hematoma. The

splint was left in place for seven days and was removed only for eating (liquid diet) and cleaning. The sutures were removed when the splint was discarded, and the wound healed uneventfully.

Four patients were not diagnosed as having a hemophilioid disease prior to operation. The development of postoperative bleeding led to the coagulation studies. One of these patients required a transfusion of whole blood before the postoperative bleeding was arrested. Pressure packs and suturing finally controlled the bleeding in the other three patients.

TABLE II. COAGULATION DATA IN NINE CASES OF HEMOPHILIOID DISEASES

CASE	TYPE OF DEFICIENCY	BLEEDING TIME (MINUTES)	COAGULATION TIME (MINUTES)	PLASMA CLOT TIME (SECONDS)	PROTHROMBIN TIME (SECONDS)	THROMBOPLASTIN GENERATION (SECONDS)	PROTHROMBIN CONSUMPTION (SECONDS)
1	PTA*	3½	6½	76	20	Plasma 21 Serum 14	15-18
2	PTC*	-	-	105	18	Plasma 10 Serum 20	16-20
3	PTC*	1½	7½	103	17	Plasma 9½ Serum 16	15½-19
4	PTC*	2	7	94	17	Plasma 10 Serum 16	18
5	PTA	2	7	82	18	Plasma 13 Serum 16	20-22
6	PTC	2½	8	105	19	Plasma 10 Serum 22	13-15
7	PTA	3	10	75	19	Plasma 14 Serum 13	17
8	PTC*	6	11	95	18	Plasma 10 Serum 17	20-31
9	PTA	5	6	115	20	Plasma 13 Serum 25	12-14

*Diagnosed preoperatively.

The diagnoses in this group of nine patients were established by the results of the thromboplastin-generation test. Results of the prothrombin-consumption test were abnormal or "borderline" in all but two of the nine patients. The results of the usual laboratory tests in all the patients studied were within normal limits, with the exception of two cases in which there were slightly prolonged bleeding times. These routine studies, along with the results of the more specific coagulation studies, are summarized in Table II. Table III defines the tests and the normal values used in the clinical pathology laboratory at the clinic.

COMMENT

As already noted, eight of the nine patients with hemophilioid abnormalities had definite personal histories of bleeding, and the other patient had a

vague but probable history of bleeding. Evidence of the condition being familial was definite in two of the patients.

The defects in five of the patients were discovered prior to operation, since their past histories concerning a prolonged bleeding tendency indicated a condition that was sufficiently serious to warrant special coagulation studies. In four of these patients, 500 ml. of whole blood was given preoperatively, whereas 250 ml. was given to the fifth patient, a 6-year-old child.

The remaining four patients, in whom the deficiency was not diagnosed preoperatively, had postoperative complications, and in all instances the postoperative bleeding was more severe, lasted longer, and was more difficult to control by the use of local measures than it was in the preoperatively diagnosed and prophylactically prepared patients. The extent of the operations performed was variable, as seen in Table I.

TABLE III. COAGULATION STUDIES USED IN DIAGNOSIS OF HEMOPHILIOID DISEASES

Bleeding Time
Time required for bleeding to stop when ear lobe is punctured; blood is blotted every 30 seconds; normal = 3 minutes
Coagulation Time
Time for 1 ml. of whole blood to gel firmly in plain glass tube at room temperature; normal = 6-10 minutes
Plasma Clot Time
Clot time at 37° C. of oxalated plasma when optimally calcified; normal = less than 100 seconds
Prothrombin Time
Clot time at 37° C. of oxalated plasma accelerated by addition of extract of rabbit brain (not acetone dried); normal = 17-19 seconds
Thromboplastin-Generation Test
Based on the method of Biggs and Douglas,[2] with adaptations of Duckert and associates[4]; normal = 9-11 seconds for both plasma and serum
Prothrombin-Consumption Test
Prothrombin time (with acetone-dried rabbit brain) of serum from blood clotted for 60 minutes at 37° C.; serum is mixed with rabbit plasma treated with tricalcium phosphate; normal = more than 20 seconds; definitely abnormal = less than 15 seconds; equivocal = 15-19 seconds

This series of cases is not large enough to allow us to compare the severity of postoperative complications in PTC and PTA deficiencies. Of the four patients with PTA deficiency, one was treated prophylactically with a preoperative transfusion of whole blood and had no postoperative bleeding; postoperative hemorrhage developed in the remaining three patients. Of the five patients with PTC deficiency, only one patient was not treated preoperatively with whole blood, and delayed postoperative hemorrhage developed in this patient.

It is obvious from the findings in these nine patients that the past medical history is extremely important in the recognition of a possible hemorrhagic disorder. In each instance, it was the history of previous bleeding that led us to

perform the special tests necessary to confirm the diagnosis. If a clotting defect is found to be caused by lack of PTC or PTA, preoperative preparation by transfusion of whole blood or plasma appears to be a justified precaution.

SUMMARY

Nine patients who had hemophilioid disorders were studied. Five patients were deficient in plasma thromboplastin component (PTC deficiency or Christmas disease), and four had an abnormality resembling deficiency of plasma thromboplastin antecedent (PTA).

Preoperative transfusion apparently was of definite help in preventing postoperative hemorrhagic complications.

The past history indicated that eight of the patients had experienced definite hemorrhagic complications during previous surgical treatment. These cases illustrate the importance of a careful preoperative history of past surgical and traumatic experiences and bleeding tendencies, both personal and familial.

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PHARMACOLOGY AND THERAPEUTICS

INVESTIGATIONS OF A NEW DENTAL CHEMOTHERAPEUTIC AGENT IN THE PRESENCE OF BLOOD

Herbert M. Cobe, Philadelphia, Pa.*

THE needs of the dental profession with respect to medicaments are unusual. Many excellent bactericides have serious shortcomings when their use in the mouth is considered. Such products as phenol have a sclerotic effect on healthy tissue. The topical usage of penicillin and other antibiotics raises the hazards of sensitization. Many other effective antibacterial agents are dangerous to use because of their toxic nature. Aqueous hydrogen peroxide continues to be widely used by the dental profession. Its lack of toxicity and the fact that it is relatively free of adverse side reactions, together with its unique combination of bactericidal action and ability to cleanse the soft tissues of debris, make it of particular value to the dentist.

Much has been written regarding the fact that aqueous peroxide in vivo loses its ability to destroy many of the same bacteria which it effectively destroys in vitro. The supposition is that this is a result of the extremely rapid breakdown of the peroxide in contact with body tissue. In this report we have attempted to determine the relationships between the speed of breakdown of the peroxide and its antibacterial action. We have also investigated a new product which, it is claimed, produces a more stable hydrogen peroxide with a more sustained action when in contact with body fluids and hence may offer a peroxide which has all the advantages of the older product without the inherent limitations.

The correlation of in vitro methods of evaluating bactericides with the in vivo performance of these agents is a constant problem. The value of in vitro tests as an indication of the effectiveness of bactericides when used in vivo is often challenged. The challenge is perhaps warranted in the case of the familiar 3 per cent aqueous hydrogen peroxide. Recent advances in preparing sources of hydrogen peroxide in which the hydrogen peroxide is broken down and oxygen is evolved over longer periods have led to a revival of interest in the use of such products. As part of a continuing study in this field, the question of a more appropriate in vitro test was considered and some preliminary work

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has been done on a radically new approach to this problem. While this technique was devised to determine the antibacterial effect of peroxides, it is believed to be applicable to numerous other antibacterial agents, especially where topical application is concerned, and it is more reliable than the inhibition techniques which measure zones of inhibition.

The action of aqueous hydrogen peroxide as a bactericide accompanies its breakdown into oxygen and water. When it is used topically (for example, in cleansing topical lesions of the skin or mucous membrane), this breakdown is greatly accelerated by the enzymes catalase or peroxidase present in most body fluids. The exudates present in lesions, especially infected lesions, of the skin or mucous membranes, as well as the blood, appear to be especially rich in these enzymes; they are present in large numbers of aerobic bacterial species but are lacking in anaerobic bacterial cells.[1-3] Hence, peroxides tend to break down very rapidly in contact with such fluids or bacteria, their bactericidal action (if any) being thus very brief. The relative ineffectiveness of aqueous hydrogen peroxide as a bactericide in vitro has usually been ascribed to this too rapid breakdown. For the same reason, attempts have been made to prepare more effective peroxides which would preserve in vivo the many good qualities of hydrogen peroxide, with emphasis toward prolonging the period during which oxygen is released and remains in contact with the tissues or infected areas.

In the many in vitro tests devised to evaluate the action of peroxides or compare them with other bactericides, this factor (the acceleration of the breakdown of peroxides by body fluids) does not appear to have been considered. Reid,[4] describing attempts to prepare peroxide ointments using polyether alcohols, attempted to measure the period over which oxygen is evolved under fixed laboratory conditions. These results were in no way correlated with bactericidal action, however; nor were they intended to simulate the approximate conditions of actual use.

Obviously, the conditions prevailing when a peroxide is applied to a contaminated or infected lesion are extremely complex. Some of the factors involved are (1) the type of enzyme present, (2) the amount of enzyme present, (3) the viscosity of the body fluid, (4) the degree to which the medicament actually comes into contact with the organisms present, and (5) the viscosity of the medicament at the surface of the lesion.

Confronted with this problem, it occurred to us that a newer approach to the problem could be made if measurements were taken when the peroxide was in contact with blood. Blood is very rich in catalase, it is relatively uniform, and it can be considered, in a measure, typical of the body fluids normally present in a large number of contaminated lesions.

CHOICE OF SUBSTRATE

In preliminary work (not reported in this article) I found that human blood plasma had no effect in causing the breakdown of the test solutions; this

is further substantiated by work previously reported by Brown and his associates,[5] who found that the addition of 10 per cent horse serum had little effect on zones of inhibition produced by urea peroxide glycerol solutions with both gram-negative and gram-positive bacteria as determined by a modified cup plate method.

It is our opinion that since blood, which is very rich in catalase, represents the body fluid which will effect the most rapid breakdown of peroxide solutions, it is the most severe reagent that can be used in these tests and could be expected to have the most adverse effect on their bactericidal action. Therefore, blood was the substrate chosen for all of the tests in the present study.

In the experiments described here, 3 per cent aqueous hydrogen peroxide is compared with a stabilized solution of urea peroxide in an anhydrous glycerin which has recently been described as being of interest in the treatment of oral lesions. Solutions of urea peroxide in glycerin were described by Brown and his co-workers[5, 7] as being more stable, longer acting, and more effective as topical bactericides. The product* under investigation represents a further advance along these lines. It contains 10 per cent urea peroxide in glycerol. In order to make the comparison with aqueous peroxide more valid, it was reduced with additional glycerol until it contained the equivalent of hydrogen peroxide on an equal volume basis. This product is referred to as urea peroxide in glycerine (3 per cent) or Gly-Oxide 3 per cent.

EXPERIMENTAL PROCEDURES

The problem presents two distinct phases for investigation: (1) a comparative study to determine the rate and degree of oxygen release when in contact with an organic substrate and (2) a comparison of the bactericidal effects of the new stabilized peroxide with a commercial aqueous peroxide of the same peroxide content by a new thin blood film technique.

Part I. Height of Foam and Release Time of Oxygen.—For this part of the study, 3 per cent Gly-Oxide and 3 per cent aqueous hydrogen peroxide were diluted with water, so that the test solutions of Gly-Oxide and hydrogen peroxide had the final dilutions of 2:1, 1:1, 1:2, 1:4, 1:8, 1:16, 1:32, and 1:64. Each of these test solutions was maintained at three temperatures—37.5° C., 25° C., and 4° C.

To 1 c.c. of the Gly-Oxide test solution placed in a serology tube was added one standard size drop of citrated human blood. The mixture was shaken on a Boerner shaker. The height of foam was measured as soon as frothing stopped, and the release time of oxygen was recorded. All tests were repeated three times and an average figure was reported. The control (aqueous hydrogen peroxide) was treated in an identical manner. It is interesting to note that when human blood plasma was added to the test solutions in place of blood, there was no oxygen release with either the 3 per cent Gly-Oxide or the aqueous peroxide. This indicated that plasma does not contain the enzymes found in

*Gly-Oxide, International Pharmaceutical Corporation, 1700 Walnut St., Philadelphia, Pennsylvania.

TABLE I. RESULTS OBTAINED WITH TWO MATERIALS IN CONTACT WITH CITRATED HUMAN BLOOD
(HEIGHT OF FOAM PRODUCED AND RELEASE TIME OF OXYGEN)

DILUTION WITH WATER	GLY-OXIDE (REDUCED WITH GLYCEROL) TO 3% HYDROGEN PEROXIDE CONTENT BY VOLUME			AQUEOUS PEROXIDE (3% HYDROGEN PEROXIDE CONTENT BY VOLUME)			
	WARM 37.5° C.	ROOM TEMPERATURE 25° C.	COLD 4° C.	WARM 37.5° C.	ROOM TEMPERATURE 25° C.	COLD 4° C.	FOAM
Undiluted	6.5 cm. 1'50"	6.0 cm. 1'50"	4.8 cm. 1'50"	5.0 cm. 1'0"	4.8 cm. 1'0"	4.5 cm. 1'0"	Height Duration
2:1	6.0 cm. 2'0"	6.0 cm. 2'0"	5.5 cm. 2'0"	4.5 cm. 1'0"	4.5 cm. 1'0"	4.0 cm. 0'50"	Height Duration
1:1	5.5 cm. 1'0"	5.0 cm. 1'50"	4.5 cm. 1'45"	4.5 cm. 0'45"	4.4 cm. 0'50"	4.2 cm. 0'50"	Height Duration
1:2	4.5 cm. 0'45"	4.1 cm. 1'0"	4.1 cm. 1'10"	4.1 cm. 0'35"	4.4 cm. 0'35"	4.2 cm. 0'45"	Height Duration
1:4	2.8 cm. 0'30"	2.5 cm. 0'45"	2.4 cm. 0'45"	2.8 cm. 0'30"	2.6 cm. 0'30"	2.5 cm. 0'35"	Height Duration
1:8	1.5 cm. 0'25"	1.4 cm. 0'35"	1.2 cm. 0'35"	1.7 cm. 0'25"	1.6 cm. 0'25"	1.5 cm. 0'30"	Height Duration
1:16	0.9 cm. 0'20"	0.8 cm. 0'25"	0.7 cm. 0'25"	1.0 cm. 0'20"	0.9 cm. 0'20"	1.0 cm. 0'25"	Height Duration
1:32	0.5 cm. 0'15"	0.5 cm. 0'20"	0.4 cm. 0'20"	0.6 cm. 0'15"	0.6 cm. 0'15"	0.6 cm. 0'15"	Height Duration
1:64	0.4 cm. 0'15"	0.4 cm. 0'5"	0.3 cm. 0'20"	0.5 cm. 0'15"	0.5 cm. 0'15"	0.4 cm. 0'15"	Height Duration

Note: All figures shown represent an average of three readings.

whole blood which catalyzes the breakdown of peroxides. The results are summarized in Table I.

Part II-A. Comparison of Bactericidal Action in Presence of Blood.—A solution of citrated human blood was inoculated so that each cubic centimeter would contain approximately 250 million bacteria per cubic centimeter. The test organism was *Staphylococcus aureus,* strain No. 209.

Bacteria were added to the blood, and measured amounts of the blood were added to previously established solutions of Gly-Oxide and hydrogen peroxide. The tubes were shaken on a Boerner shaker for one minute and subcultures were taken—that is, two loopfuls were removed and spread on the surface of a blood agar plate. This was repeated two minutes from the time of mixture. (From previous experiments, it had been found that the foaming had reached the maximum before two minutes had elapsed.) Results are summarized in Table II-A.

Part II-B. The Thin Blood Technique.—A solution of citrated human blood was inoculated with *Staphylococcus aureus,* government strain No. 209, so that the final bacterial concentration was equal to 250 million per cubic centimeter. To accomplish this, (1) a physiologic saline solution containing 500 million *Staph. aureus* per cubic centimeter was prepared by the nephelometer method, and (2) citrated human blood was added to an equal part of the saline solution of *Staph. aureus,* resulting in the desired concentration of 250 million bacteria per cubic centimeter.

Commercial 3 per cent aqueous hydrogen peroxide and 3 per cent Gly-Oxide were tested undiluted and by dilution of the test material with sterile water so that the 3 per cent Gly-Oxide and aqueous hydrogen peroxide had final dilutions of 1:1, 1:2, and 1:3. Each test solution was maintained at three temperatures—37.5° C., 25° C., and 4° C.

TABLE II-A. COMPARISON OF BACTERICIDAL ACTION IN PRESENCE OF BLOOD FOLLOWING MECHANICAL AGITATIONS

	GLY-OXIDE 3%			AQUEOUS PEROXIDE 3%		
	TEMPERATURE OF TEST SOLUTION			TEMPERATURE OF TEST SOLUTION		
DILUTION H_2O	WARM 37.5° C.	ROOM 25° C.	COLD 4° C.	WARM 37.5° C.	ROOM 25° C.	COLD 4° C.
Undiluted						
1 minute	0	0	0	+3	+3	+4
2 minutes	0	0	0	+4	+4	+4
2:1						
1 minute	0	0	0	+4	+4	+4
2 minutes	0	0	0	+4	+4	+4
1:1						
1 minute	0	0	0	+4	+4	+4
2 minutes	0	0	0	+4	+4	+4
1:2						
1 minute	0	+3	0	+4	+3	+4
2 minutes	0	0	0	+4	+3	+4
1:4						
1 minute	0	0	+1	+3	+3	+4
2 minutes	0	0	0	+3	+3	+4
1:8						
1 minute	+3	+2	+2	+4	+4	+4
2 minutes	+3	+1	+1	+4	+4	+4
1:16						
1 minute	+3	+3	+3	+4	+4	+4
2 minutes	+3	+3	+2	+4	+4	+4
1:32						
1 minute	+3	+3	+3	+4	+4	+4
2 minutes	+3	+3	+3	+4	+4	+4

Code:

0 = No growth. +1 = Slight growth. +2 = Growth.
+3 = Heavy growth. +4 = Maximum growth.

All tests were made with whole human blood-saline solution and *Staphylococcus aureus* strain No. 209 (2.5×10^8 organisms per cubic centimeter).

Control showed heavy or maximum growth (+4) in all cases.

All results are based on three trials.

Two drops of the inoculated blood were placed on a slide and were spread with another slide into a thin film. Both slides used were sterile.

The slide with the thin blood film was placed in a sterile Petri dish, and 1 c.c. of test solution was added to the slide after it was ascertained that the film was uniformly covered. The control aqueous hydrogen peroxide) was treated in an identical manner. One loopful of the mixture was removed at one-minute intervals for a total of twenty minutes, and each sample was streaked on the surface of a blood agar plate. The results are summarized in Table II-B.

TABLE II-B. THIN BLOOD FILM TECHNIQUE USING STAPHYLOCOCCUS AUREUS, STRAIN NO. 209 (2.5×10^8) ORGANISMS PER CUBIC CENTIMETER) AND WHOLE HUMAN BLOOD WITH SALINE SOLUTION

TIME (MINUTES)	3% GLY-OXIDE UNDILUTED			3% HYDROGEN PEROXIDE UNDILUTED		
	WARM 37.5° C.	ROOM TEMPERATURE 25° C.	COLD 4° C.	WARM 37.5° C.	ROOM TEMPERATURE 25° C.	COLD 4° C.
1	+3	+3	+3	+4	+4	+4
2	+3	+2	+3	+4	+4	+4
3	+2	+1	+1	+4	+4	+4
4	+2	+1	+1	+4	+4	+4
5	0	0	0	+4	+4	+4
6	0	+1	0	+4	+4	+4
7	0	0	0	+4	+4	+4
8	0	0	0	+4	+4	+4
9	0	0	0	+4	+4	+4
10	0	0	0	+4	+4	+4
11	0	0	0	+4	+4	+4
12	0	0	0	+4	+4	+4
13	0	0	0	+4	+4	+4
14	0	0	0	+4	+4	+4
15	0	0	0	+4	+4	+4
16	0	0	0	+4	+4	+4
17	0	0	0	+4	+4	+4
18	0	0	0	+4	+4	+4
19	0	0	0	+4	+4	+4
20	0	0	0	+4	+4	+4

Code:

0 = No growth. +1 = Slight growth. +2 = Growth.
+3 = Heavy growth. +4 = Maximum growth.

Control showed maximum growth (+4) in all cases.
All other tests (solutions 1:1, 1:2, 1:3, etc.) showed maximum growth.

RESULTS

Part I of the present study reveals the following:

1. Gly-Oxide has a slower rate of reaction with citrated human blood than does aqueous hydrogen peroxide, especially at room and cold temperatures and in the concentrated solutions. Aqueous hydrogen peroxide in contact with blood breaks down immediately and to a greater extent than Gly-Oxide. Hydrogen peroxide in warm concentrated solutions lacks stability, even when not in contact with blood, partially losing its contained oxygen. Gly-Oxide remains stable under similar conditions.
2. Increasing the temperature increases the rate of reaction of both Gly-Oxide and aqueous hydrogen peroxide.
3. The greatest effects of dilution on the rate and extent of reaction of Gly-Oxide with citrated human blood occur through the 1:8 dilution; corresponding effects of dilution on the reactions of aqueous hydrogen peroxide occur through the 1:4 dilution.

Part II-A of the study reveals the following:

1. Subcultures from the undiluted Gly-Oxide and the dilutions through 1:4 were negative for bacterial growth.

2. Subcultures from the undiluted aqueous hydrogen peroxide and all dilutions of aqueous hydrogen peroxide showed heavy or maximum growth in all cases.
3. Subcultures from the control show heavy or maximum growth in all cases.

Part II-B reveals the following:

1. Subcultures made from the undiluted Gly-Oxide were positive for growth at one, two, three, and four minutes. Subcultures made at five minutes and the following samples up to twenty minutes showed no evidence of growth.
2. Subcultures made from the undiluted 3 per cent hydrogen peroxide (aqueous) showed heavy or maximum growth in all samples taken. There was no evidence of bactericidal or bacteriostatic effect, even after a twenty-minute contact exposure.
3. Subcultures from the control showed heavy or maximum growth in all cases.
4. Gly-Oxide solutions diluted with water 1:1, 1:2, and 1:3 showed growth in all samples up to twenty minutes.

These results are summarized in Table II-B.

DISCUSSION

In preliminary attempts to measure bactericidal action in the presence of thin blood films, some studies were made of the rate of breakdown and release of oxygen from two materials in the presence of blood. Qualitative experiments demonstrated that there was a significant difference between the characters of the oxygen evolution between the two products. In the case of aqueous 3 per cent hydrogen peroxide, the evolution of oxygen is immediate; bubbles formed enlarge rapidly and tend to escape, and the opaque white appearance engendered by the oxygen bubbles disappears relatively fast. In the case of the 3 per cent Gly-Oxide the initiation of oxygen release, although prompt, appears somewhat more gradual; the foam appeared to consist of more minute bubbles, and the white opaque appearance lasted much longer.

Undoubtedly, the character and persistence of the respective foams are a complex result of a number of factors. The greater viscosity of the glycerin—or, more specifically, the glycerin solution of blood fluids which the hygroscopic glycerin slowly absorbs—plays a part. Likewise the surface tension of these mixtures and, finally, the rate of breakdown of the urea peroxide in comparison with aqueous hydrogen peroxide are also factors.

On the basis of these qualitative observations, a technique which gave some measurement of rate of release of oxygen and height of foam formed was devised. If the time of oxygen release is brief, it might be anticipated that bactericidal action might be negligible. The longer the period of oxygen release, the greater the chance for bactericidal action to take place. Also, since this oxidative bactericidal action must take place either within the liquid or

more probably at the interface between body fluid and oxygen-producing medicament, the volume of foam might also be indicative of the effectiveness of the two fluids.

Previous work[6] indicated that although saliva catalyzes the breakdown of urea peroxide solutions in both glycerol and propylene glycol, it does so to a lesser extent than is the case when pus or blood is present. It was noted that saliva catalyzed the breakdown of aqueous peroxide to a much greater extent than with the polyhydric alcohol solutions of urea peroxide.[7]

CONCLUSIONS

1. A method of measuring the effectiveness of 3 per cent aqueous hydrogen peroxide as a bactericide when brought into contact with thin blood films inoculated with *Staphylococcus aureus* is described.

2. The results confirm clinical observations that the good bactericidal action of 3 per cent aqueous hydrogen peroxide, previously demonstrated in vitro, is lost almost immediately when the material is brought into contact with such films and that this is due to the rapid breakdown of the peroxide accompanied by the rapid evolution of oxygen caused by the enzymes present in the blood films.

3. A preparation of urea peroxide in glycerin of comparable hydrogen peroxide content in the presence of such thin blood films breaks down more slowly and retains the oxygen in contact with the blood as a compact foam. During an appreciable period, significant bactericidal action is demonstrated, especially where the glycerin solution is used without previous water dilution.

4. The more prolonged evolution of oxygen, the character and persistence of the resultant oxygenated foam, and the time during which bactericidal action remains effective lend support to clinical observations previously reported[7] that the cleansing antibacterial action long ascribed to 3 per cent aqueous hydrogen peroxide is lost in the presence of thin blood films, and probably in the presence of other enzyme-rich body fluids, but is realized in worthwhile measure by the more stable, slower reacting urea peroxide-glycerol solution.

5. Bacteriologic results suggest that the urea peroxide solution in glycerol, when employed in the presence of blood, should be used undiluted or in minimum dilution to achieve maximum antibacterial action.

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ANESTHESIOLOGY

A NEW INTRAVENOUS BARBITURATE FOR PEDIATRIC ANESTHESIA IN DIFFICULT AND HANDICAPPED DENTAL PATIENTS

Max H. Douglas, D.D.S., Hempstead, N. Y.*

INTRAVENOUS anesthesia in children plays a far more important role than is generally conceded. To date, thiopental sodium has superseded almost entirely the other intravenous barbiturates, but it should seldom, if ever, be used alone. It is best used in moderate doses in combination with other agents. It fortifies such agents as nitrous oxide–oxygen and permits the more toxic agents to be given in smaller amounts. It quiets breathing and prevents a great deal of postoperative nausea and vomiting, even after ether anesthesia.

The use of intravenous agents for general anesthesia in ambulatory patients is increasing. Induction is usually pleasant and rapid, and there is a minimum of nausea and vomiting. However, for the prolonged anesthesia needed for the complete dental treatment in one visit of difficult and handicapped children, it was found that a prolonged recovery time was the main drawback of the use of thiopental sodium.

This is not a serious problem in cases that are treated in the hospital. In our practice, however, almost 100 per cent of our work is done in our offices on ambulatory patients, and prolonged recovery time is quite a serious disadvantage.

We have tried a variety of other intravenous barbiturates in an effort to overcome this problem, but without success. Methitural sodium seemed very promising and was eagerly added to our armamentarium, but it proved to be a very great disappointment. The recovery period is shorter with methitural sodium than with the other barbiturates, but only when the anesthetic procedure is very short, as in minor oral surgery. For prolonged anesthesia, however, the accumulation of methitural sodium in the body tissues makes for as prolonged a recovery time as that which follows any of the intravenous so-called "short-acting" barbiturates.

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Several months ago Eli Lilly and Company supplied us with a new ultra-short-acting intravenous barbiturate, methohexital. The findings in ninety-five cases treated in our offices, with the use of methohexital as the only change in our routine, are reported here.

Methohexital sodium differs chemically from all other barbiturate anesthetics in that it contains no sulfur in the molecule. Chemically, it is sodium 1-methyl-5-allyl-5 (methyl-2 pentynyl) barbiturate. It is stable in aqueous solution at room temperature (25° C.) for at least six weeks. Like other sodium barbiturate salts, such solutions have a pH of approximately 11.0. Solutions of methohexital sodium should not be mixed with acid solutions, such as atropine sulfate and succinyl choline chloride.

Compared with other intravenous anesthetic barbiturates, methohexital sodium exhibits (1) greater potency (two to five times), (2) shorter duration of effect (one-half as long), and (3) more prompt recovery, even after large doses.

In all animal studies of potency and duration of action of single doses, methohexital proved twice as potent as thiopental. Its action was characterized by a rapid and smooth induction, satisfactory surgical anesthesia, and quick recovery.

On repeated doses, methohexital is less cumulative than the thiobarbiturates.

The plasma concentration of methohexital during anesthesia is approximately one-half that of the thiobarbiturates, indicating that methohexital is twice as potent. During recovery, blood concentrations decreased rapidly. After twenty-four hours, no methohexital could be identified in the blood, whereas blood levels of the thiobarbiturates were approximately 35 per cent of the levels present at the end of the third hour of anesthesia. This shows that methohexital is more rapidly metabolized.

There are no significant changes in blood pressure and respiration in prolonged anesthesia with methohexital in the usual doses (10 mg.), and there is no measurable impairment of renal or hepatic function.

Methohexital sodium may be used with any of the recognized preanesthetic medications. It may also be used with inhalation anesthetics, skeletal-muscle relaxants, and other medications. No drug is known to lessen its effectiveness. There are no known clinical conditions in which the use of methohexital is specifically contraindicated. When general anesthesia is contraindicated, however, methohexital should not be used.

It is recommended that methohexital be used in a 1 per cent solution (10 mg. per c.c.). It may be diluted with normal saline, water for injection, or 5 per cent dextrose in water for intravenous use.

In our practice each patient has an initial appointment, at which time a complete history is taken and the child's mouth is examined to determine the extent of the dental services required (Figs. 1 and 2). The parents are then acquainted with as many of the significant details of the anesthetic procedure as it is deemed desirable for them to know. Also, at this time the child's pulse, blood pressure, and respiratory rate are recorded.

A prescription for preanesthetic medication consisting of promethazine hydrochloride, secobarbital, and atropine sulfate (dosages depending on the age, weight, and development of the child) is given the parent with full instructions

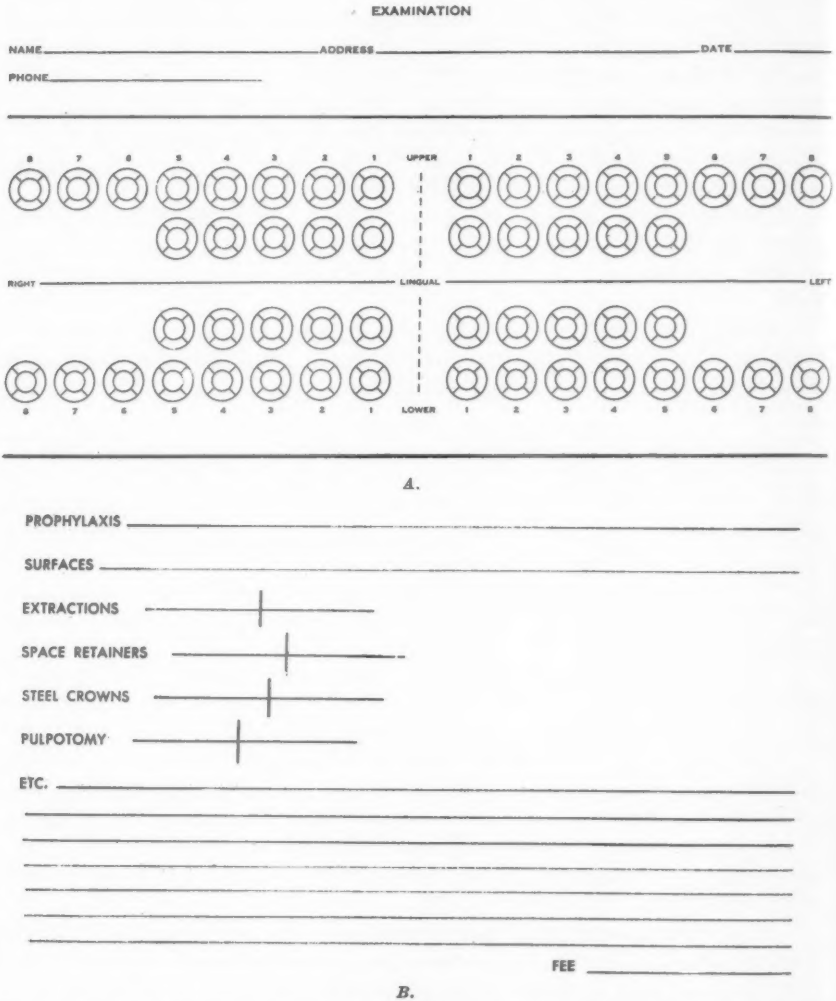

Fig. 1.—Form used for recording results of oral examination *(A)* and extent of dental services required *(B)*.

for administration. The parent is instructed to take the child to a physician for an anesthetic evaluation and to bring back a full report of his findings along with a report of a complete blood count and urinalysis. An appointment is then made for the anesthetic and dental procedures.

On the morning of the operation the child is brought to the office in pajamas. This eliminates the time-consuming task of undressing the patient and also reduces the amount of handling and the resultant disturbance which it may cause.

CONSULTATION

_____ 19 ____

PATIENT

NAME _____ AGE _____ BIRTH DATE _____

ADDRESS _____ FATHERS NAME _____

BUS. ADDRESS _____

PHONE _____ CONSULTATION FEE _____ BUS. PHONE _____

COMPLAINT _____

TYPE OF PATIENT

UNMANAGEABLE _____ CEREBRAL PALSY _____

MENTALLY RETARDED _____ DESIRES THIS TYPE OF TREATMENT _____

OTHER _____

X-RAYS RECENT _____ OLD _____ TAKEN _____ CLINICAL EXAM _____

REFERRING DENTIST

NAME _____

ADDRESS _____

PHONE _____ LETTER ☐

PATIENT'S PHYSICIAN

NAME _____

ADDRESS _____

PHONE _____ LETTER ☐

COMMENTS _____

MEDICAL HISTORY

1. SERIOUS ILLNESSES _____
   - A) HEART _____
   - B) LUNGS _____
   - C) KIDNEYS _____
   - D) BLOOD _____
   - E) RHEUMATIC FEVER _____
2. UNDER TREATMENT BY PHYSICIAN _____
3. TAKING ANY MEDICATION NOW _____
4. PREVIOUS GENERAL ANESTHETIC _____

SERVICE DESIRED _____

PERMISSION FOR OPERATION DATE _____ FEE _____

I HEREBY AUTHORIZE DR DOUGLAS TO PERFORM ANY PROCEDURES OR OPERATIONS UPON ME _____ AND TO ADMINISTER SUCH ANESTHETICS, WHICH IN THE JUDGEMENT MAY BE NECESSARY OR ADVISABLE WHILE I AM _____ UNDERGOING THESE PROCEDURES IN THE OFFICE

SIGNATURE _____ RELATIONSHIP _____

_____
WITNESS

Fig. 2.

The child is placed on the operating table, a stethoscope is taped over the left chest to record the heart beats, and a blood pressure cuff is applied to the right arm to record the blood pressure intermittently.

Anesthesia is then started by gradually allowing an 80:20 mixture of nitrous oxide–oxygen to be wafted over the child's face until he is relaxed, at which point the mask is placed on the face. The non-rebreathing technique used throughout offers the following advantages:

1. It allows for rapid removal of nitrogen from the lungs, permitting higher alveolar concentration of weaker anesthetic agents.
2. It allows for a higher concentration of oxygen in the lungs.
3. It provides adequate carbon dioxide elmination.
4. It dissipates excessive heat from the lungs, thus helping to prevent a rise in body temperature.

As soon as the patient begins to lose the lid reflex, a venipuncture is made (usually in the left arm, if a vein is palpable, or in the left saphenous vein in the ankle). A 5 per cent dextrose drip is started, mainly to keep the vein open for intravenous medication and also to prevent a certain amount of dehydration, since the patient has ingested nothing by mouth since dinner the night before.

Methohexital, 30 mg., is now injected into the drip cuff, the nitrous oxide–oxygen mixture is changed to 50:50, and the Trilene vaporizer is opened gradually to # 2. At this point it is usually necessary to administer an additional 20 mg. of methohexital to relax the child so that the mouth can be opened and the pharynx examined with a laryngoscope without initiating a spasm. All secretions are now suctioned out, and the pharynx and glottis are sprayed with a topical anesthetic.

As soon as a proper endotracheal tube is selected and lubricated with a water-soluble anesthetic jelly, 20 mg. of succinyl choline chloride is injected into the intravenous cuff, and the child is allowed to breathe 100 per cent oxygen until all respirations cease. The tube is inserted nasally (very carefully so as not to tear the membranes and cause bleeding), the mouth is opened and, by means of a laryngoscope, the larynx is exposed and the tube is inserted past the vocal cords with the aid of a Magill forceps.

A Fink valve and bag is connected immediately to the tube, and the lungs are inflated with 100 per cent oxygen. The tube is then taped to the face to prevent slipping, and the child's head is placed in a "doughnut" made up for the purpose of stabilizing the head. A mouth prop is placed between the teeth on one side, the pharynx is examined carefully and suctioned out again if necessary, and a moistened throat pack is carefully placed around the endotracheal tube and back of the tongue.

By this time, the effects of the succinyl choline chloride are wearing off and spontaneous respirations have begun. As soon as the respirations become rhythmic, the anesthetic mixture is reset to nitrous oxide–oxygen 50:50 and Trilene # 2½ on the vaporizer. If there is a slight bucking on the tube, indicating a lightening of the anesthetic level, 20 mg. of methohexital is again injected intravenously.

The patient is now ready for the dental procedures to begin. If preoperative roentgenograms were not obtained earlier, the child's teeth are now x-rayed with a fast film. The films are developed immediately to aid us in our dental procedures. The teeth are thoroughly cleaned at this time; the cleaning material is flushed out and the mouth is suctioned out very thoroughly.

By means of ultra-high-speed dental equipment and a steady stream of water, the teeth are prepared for the necessary fillings and/or crowns in a very

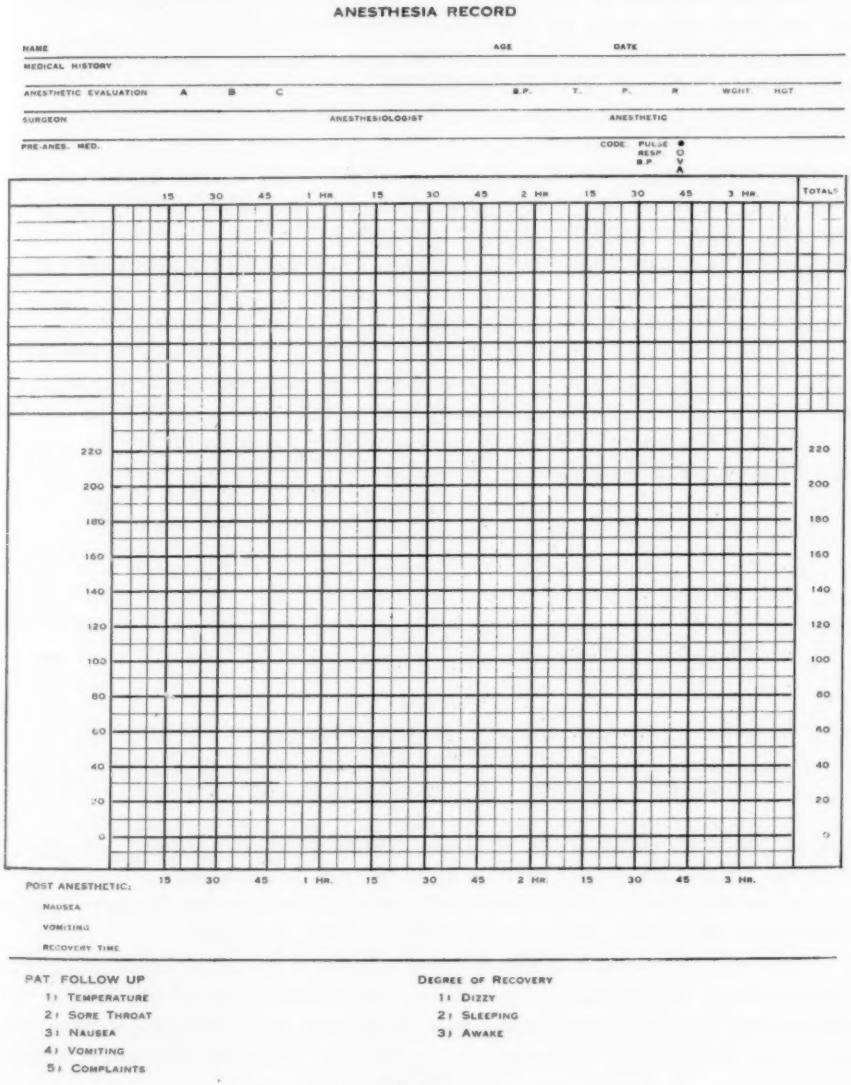

Fig. 3.

short time. Exposed pulps are capped or pulpotomies are performed, depending on the judgment of the operator. When teeth must be removed, space retainers may be constructed and cemented. (We use the Rocky Mountain crowns and/or bands and loops prepared with the orthodontic welding machine.)

When treatment of one half of the mouth is completed, the area is flushed and suctioned out, the bite block is shifted, and the other half of the mouth is prepared and treated. Bands and wedges are used on all interproximal fillings. All amalgam restorations carved, and the occlusion is adjusted by removing the bite block and testing the lower teeth against the upper teeth.

All this time, of course, the anesthetist is busy checking and recording the pulse, blood pressure, and respirations at five- to ten-minute intervals and generally attending to the patient's needs (Fig. 3). As the anesthetic effects of the methohexital lighten, more is injected in 10 to 20 mg. doses as the case requires.

When the dental procedure has been completed, the mouth is thoroughly cleaned of all debris, the throat pack is removed carefully, the Trilene and nitrous oxide are discontinued, and the patient is allowed to breathe pure oxygen. It is usually found that in less than five minutes the patient is beginning to buck on the tube. As soon as the tube becomes too intolerable to the patient, it is removed and a face mask is applied so that the child may breathe pure oxygen for a little while longer. At this point the patient usually becomes rather restless and begins to cry and toss about. The intravenous dextrose is then stopped, the child is turned on his belly, and a little more oxygen is given by face mask. When the child begins to react a little more to his surroundings and shows signs of more reflexes returning, he is taken to the recovery room where one of the nurses will stay in attendance. The room is equipped with suction and oxygen, should they become necessary.

Usually the child has all of his reflexes back within fifteen minutes and, as a rule, is ready to go home after thirty minutes. He may be sleepy, but he is sufficiently alert to be allowed to go home after the parents have been properly instructed in postanesthetic care.

TABLE I

	AGE (YEARS)										
	2	3	4	5	6	7	8	9	10	11	12
Number of patients	10	25	25	10	8	1	1	8	0	2	5

Of the ninety-five cases reported, the ages ranged from 2 years to 12 years, with the greatest number of patients being between 3 and 6 years of age (Table I). There was an equal distribution of the sexes. The patients' weights ranged from 26 to 104 pounds, with a mean weight of about 45 pounds.

The initial dose of methohexital was generally 50 mg. (Tables IIA and IIB). The 2- and 3-year-old patients, as well as those children who were very well relaxed by the premedication, usually received an initial dose of 20 to 30 mg. The total dose of methohexital ranged from 30 mg. to 250 mg., with an

average dose of 130 mg. The anesthetic period varied from thirty minutes to three hours, with the largest number of anesthesias lasting one and one-half hours (Table III).

In three of the first several patients treated after methohexital was first used, there was some jactitation and twitching. Tremors were seen in two patients, hiccupping occurred in two patients. This reaction was attributed to a too-rapid injection of the methohexital. There have been no such effects when the drug has been injected very slowly.

TABLE II A

	INITIAL DOSE OF METHOHEXITAL (MG.)		
	20	30	50
Number of cases	14	24	57

TABLE II B

	TOTAL DOSE OF METHOHEXITAL (MG.)																		
	30	50	60	70	80	90	100	110	120	130	140	150	160	170	180	190	200	220	250
Number of cases	2	6	1	4	2	4	12	9	10	3	10	6	4	9	1	1	5	2	4

Methohexital has been found to be rapidly effective, and the patients are readily maintained at the desired plane of anesthesia with small additive doses, usually 20 to 30 mg.

Postanesthetic nausea and vomiting have not been observed in any of our cases. Of course, there is no doubt that the preanesthetic use of promethazine hydrochloride may have some bearing on this result, although with the thiobarbiturates a fair precentage of postanesthetic nausea was experienced and we routinely injected each patient with 25 to 50 mg. of Marezine about thirty minutes after he was anesthetized.

TABLE III

	DURATION OF ANESTHESIA (HOURS)					
	½	1	1½	2	2½	3
Number of cases	8	25	36	8	13	5

We called the parents of each patient at home about five to six hours after the patient was discharged from the office. Fifty per cent of the parents reported that their children went back to sleep and slept for three to four hours. The other half reported that their children were either completely alert by the time they got home or within the hour were up and asking for food. Less than 1 per cent reported any nausea and vomiting. There were no reports of headaches, and only very few reported some slight dizziness which lasted for a short time.

SUMMARY

1. The use of a new intravenous barbiturate for pediatric anesthesia has been described.

2. In ninety-five unselected cases this anesthetic was used very successfully in children 2 to 12 years of age.

3. There were no clinical signs of side reactions except as noted, and these were caused by a too-rapid injection of the initial dose.

4. There was only a mild rise in blood pressure as against a greater rise with the thiobarbiturates.

5. There was no coughing or laryngospasm in any of the cases reported.

6. There was no depression of respiration in any case in the doses used.

7. Recovery was rapid, usually within fifteen minutes, and all patients were permitted to leave the office at the end of thirty minutes.

Methohexital sodium was supplied by Dr. Charles M. Gruber, Jr., of the Lilly Laboratory for Clinical Research, Indianapolis General Hospital, Indianapolis, Indiana.

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544 FRONT ST.

CONSULTANT'S COMMENT (HAROLD W. KROGH)

Dr. Douglas has written an interesting paper on pediatric general anesthesia, but the title is misleading. This is not a description of intravenous barbiturate anesthesia. In fact, methohexital sodium is not even the principal drug; nitrous oxide–oxygen should be given top billing.

The technique described, while no doubt effective in the hands of Dr. Douglas, sounds to me like balanced anesthesia gone berserk.

A quick reading of the paper reveals that promethazine hydrochloride, secobarbital, atropine sulfate, nitrous oxide, methohexital, succinyl choline chloride, a topical anesthetic, and oxygen were given to a single patient.

EDITORIAL COMMENT (DANIEL J. HOLLAND)

The technique and multiple use of drugs outlined in Dr. Douglas' paper provide an excellent example of the need occasionally to reflect and to analyze critically. Dr. Krogh has repeatedly stated that he is unconvinced that "many poisons are better than one." When as many as eight drugs are used to produce a state of general anesthesia, one cannot help but wonder about the

pharmacologic disposition of each drug employed, the complexity of detoxification and elimination, the "beef-stew" effect of multiple side products, and chain reactions. The old adage about simplifying the complex rather than complicating the simple seems to apply here. It is difficult to comprehend any balance physiologically in this type of so-called balanced anesthesia.

The never-ending array of new pharmaceuticals lavishly advertised, samples of which are generously supplied, presents difficult professional temptations. Is it unreasonable to expect the doctor to know the basic chemistry of the drug or drugs used, to trace distribution and elimination whether the drug be inhaled, injected, or swallowed, and to be familiar with breakdown end products and their pharmacologic influences? Does common practice of administration and manufacture by a reputable firm satisfy the professional conscience and meet legal requirements? Or are we being too picayune?

ORAL ROENTGENOLOGY

American Academy of Oral Roentgenology

Arthur H. Wuehrmann, Editor

RADIATION DOSAGE IN DENTAL OFFICES

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TWO HUNDRED dental x-ray installations in the Boston area have been surveyed during the past year to ascertain levels of radiation dosage currently received by patients and operators during dental x-ray examinations in private dental offices. The surveys were made possible through the cooperation of practicing dentists and were carried out by specially trained teams of dental students. Preliminary questionnaires returned by the participating dentists were followed up by office surveys for which standard radiation-monitoring instruments were used. Following the compilation and analysis of the data for each installation, the dentists were mailed reports of the individual dosages recorded, together with specific recommendations by which these dosages could be reduced to minimum levels in each case.

METHOD OF PROCEDURE

Dentists selected at random within the metropolitan area were invited by letter to participate in the survey, and the responses received covered 200 x-ray installations, all of which are represented in the findings comprising this report. The content of the questionnaire accompanying each letter was similar to that devised by Yale and Hauptfuehrer,[6] with additional information being sought in the area of film exposure and processing techniques. The completed questionnaires were followed by site visits, when an opportunity was taken to make objective checks of the responses to certain of the questionnaire items.

This study was supported by United States Public Health Service Grant D-958.
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During each office visit the primary dosage for an upper incisor film exposure was monitored in air at cone-tip distance with a Victoreen #70 or #570 condenser r meter, using a standardized beam projection on to a film positioned in the dentist's mouth behind the incisor teeth. During this intraoral test exposure, scatter radiation was monitored 3½ feet above floor level at the normal operating location by means of a Nuclear-Chicago #2586 survey meter. The primary dosage was also measured outside the mouth by a second exposure made on special dosimetric film at cone-tip distance. Simultaneously, a 5 by 7 inch no-screen film was exposed beneath the dosimeter film to obtain an objective record of the dimensions of the primary beam at cone-tip distance.

The operating factors customarily used by the dentist were recorded, together with the brand and model of the x-ray equipment, its kilovolt peak and milliamperage ratings and controls, the measurements of any added filtration or added collimation, and the result of a five-second timer check made with a stopwatch. Unexposed samples of films used by the dentist were obtained for positive identification of film type. All the information obtained from the questionnaire and from the office survey was subsequently coded for entry on keysort cards to facilitate analysis of the data.

CHARACTERISTICS OF THE SAMPLE

The dental x-ray equipment that was surveyed included a good representation of the most commonly used brands and models, together with a few lesser known units and six of the latest 90 KVP models. Table I lists the equipment by brand and model, together with the best currently available information on their standard ratings and operating factors as delivered by the manufacturers. Table II indicates the frequency and extent of modifications to these standard features, as observed during the site visits. This tabulation reveals that few dentists were making use of two equipment modifications which can facilitate the use of faster films with late-model x-ray machines whose spring-operated timing devices are ineffective for fractional-second exposures. Only 8.5 per cent were substantially reducing the milliamperage below the almost universal 10 Ma., and only 1.5 per cent of the x-ray units were operated with an extended anode-to-film distance.

In 36.5 per cent of the installations a standard beam diameter at cone-tip of 3 inches or less was used, but an additional 15.5 per cent were found to be carrying supplemental collimation washers to achieve this result or better. This leaves a high percentage (48.0 per cent) being operated with a beam diameter exceeding 3 inches, almost entirely made up of units with beam diameters of either 4.0 or 5.5 inches. It was found that 21.5 per cent of the machines had a standard internal or built-in filtration equivalent in total to 1.5 mm. aluminum, and an additional 41.0 per cent were fitted with supplementary filter disks to achieve at least that level of filtration. However, 37.5 per cent of the units were being operated with less than the 1.5 mm. aluminum equivalent total filtration recommended in the current *Handbook #60* of the National Bureau of Standards.[4]

In 31 instances, units were operated from behind some kind of partition or screen. In the other 169 situations relatively poor advantage was being taken of the protection available to the operator through distance from the patient. Forty units with 5 foot timer cords and 104 units with 6 foot timer cords were alike operated at an average distance of 4.6 feet from the patient's head, but twenty-six units with timer cords of from 7 to 11 feet were operated at an average distance of only 5.6 feet, and twenty-seven units with timer cords of 12 feet or longer were operated at an average distance of only 6.1 feet.

TABLE I. SAMPLE CHARACTERISTICS
STANDARD FEATURES OF X-RAY EQUIPMENT SURVEYED IN DENTAL OFFICES

NUMBER OF UNITS SURVEYED	EQUIPMENT LISTED BY BRANDS AND MODELS	RATED KVP	AVAILABLE MILLI-AMPERAGE	STANDARD ANODE-TO-CONE-TIP DISTANCE (INCHES)	STANDARD BEAM DIAMETER AT CONE TIP (INCHES)	RATED INTERNAL FILTRATION (MM. ALUMINUM EQUIVALENT)
1	Fischer (open tube)				4.00	0.50?
5	Fischer (closed head)		5-15		4.00	0.50?
34	General Electric CDX	65	10	8.00	3.00	1.40
5	General Electric 70	70	10	8.00	3.00	1.50
5	General Electric 90	50-90	10, 15	8.00	3.00	1.50
9	Philips Oralix	45	5	4.00	2.50	1.00
1	Prof-Ex-Ray		5-20		4.00	0.50?
11	Ritter Model A	45?	10		4.00	0.50
47	Ritter Models B, C	63	10	7.00	4.00	0.50
16	Ritter Model D	50-70	5-15	7.00	4.00	0.50
20	Victor	55	10		3.00	0.75
23	Weber (to Model 11)	45-70	5-15	6.25	4.00	0.50
1	Weber Model 12	50-90	1-20		3.50	0.50?
22	XRM Models 1-3, to 1956	50-70	7-20	5.50	5.50	0.75
200						

Sources for this tabulation are data from this survey; data published by A. G. Richards (J. Am. Dent. A. 54: 476, 1957); and information supplied by manufacturers of the equipment.

Relative speeds for the films used with these units are indicated in Table III. It was found that 52 per cent of the machines were operated in conjunction with the use of either slow or medium-speed films, despite the potential major contribution of fast film speeds toward dosage reduction. This finding cannot be related to any generally valid objection to the diagnostic qualities characteristic of faster films; it may, rather, reflect widespread misinformation on the relative speeds of various film types. In support of this inference, we found that only 60 per cent of the dentists were able to classify their films correctly in terms of slow, medium, or fast speeds. Many of the

remaining 40 per cent were users of a well-known medium-speed film, and almost without exception, they classified it as a fast film. Was this because it has long been blessed with a fast-sounding name? Or have dentists been misled by promotional publicity which has claimed—correctly enough—that the film's speed has been increased within recent years? The fact is that after allowance has been made for the increased speed, this film still qualifies as only medium-speed, even as related to the former sensitivity of fast films that have long since been further increased in their emulsion speed.

TABLE II. SAMPLE CHARACTERISTICS
MODIFICATIONS TO X-RAY EQUIPMENT SURVEYED IN DENTAL OFFICES

NUMBER OF UNITS SURVEYED	EQUIPMENT LISTED BY BRANDS AND MODELS, GROUPED BY OPERATING KILOVOLTAGES	OPERATING MILLIAMPERAGE OTHER THAN 10 MA. (±1)		EXTENDED ANODE-TO-CONE-TIP DISTANCE (10 IN. OR MORE)	WITH ADDED BEAM COLLIMATION		WITH ADDED BEAM FILTRATION	
		12 MA. OR OVER	8 MA. OR LESS		NO. OF UNITS	MEAN BEAM DIAMETER (INCHES)	NO. OF UNITS	MEAN TOTAL FILTRATION (MM. ALUMINUM)
	Operated at 45 to 55 KVP							
9	Philips Oralix	–	9	–	–	–	–	–
11	Ritter Model A	–	–	–	1	2.5	3	2.00
1	Ritter Model B	–	–	–	–	–	–	–
20	Victor	7	1	–	–	–	6	1.83
1	Weber Model 11	–	–	–	–	–	–	–
2	XRM Models 1-3	–	–	–	–	–	–	–
44								
	Operated at 60 to 70 KVP							
34	General Electric CDX	2	–	–	–	–	11	2.35
5	General Electric 70	–	–	–	–	–	–	–
62	Ritter Models B-D	9	4	3	16	2.7	29	1.60
22	Weber Models to #11	4	1	–	9	2.6	12	2.10
1	Weber Model 12	–	–	–	–	–	1	2.00
20	XRM Models 1-3	5	1	–	4	2.7	12	1.96
144								
	Operated at 90 KVP							
5	General Electric 90	5	–	–	–	–	5	2.30
	KVP not determined							
1	Fischer (open tube)	–	–	–	–	–	1	1.00?
5	Fischer (closed head)	2	1	–	1	2.8	2	2.50?
1	Prof-Ex-Ray	1	–	–	–	–	–	–
7								
200	Totals	35	17	3	31	2.7	82	1.92

Film-processing technique, the subject of Table IV, can be a significant if not a major factor in minimizing x-ray dosage through emphasis on extended development times. Films from less than 10 per cent of the units were assured of development for five minutes or longer at 68° F.; it may come as something of a shock to find visual inspection the determining factor in 46 per cent of the instances. This custom, found to be equally prevalent among older dentists

TABLE III. SAMPLE CHARACTERISTICS
DENTAL X-RAY FILMS USED WITH THE SURVEYED X-RAY INSTALLATIONS

APPROXIMATE RELATIVE EMULSION SPEED*	BRANDS OF FILM, GROUPED BY EMULSION SPEED	MANUFACTURERS' FILM DESIGNATIONS	NUMBER OF X-RAY UNITS WITH WHICH FILMS ARE USED (N-200)
1	*Slow Films*		
	DuPont	551 "S"	1
	Eastman Kodak	"Regular"	0
	I.F.I.	"Intermediate"	7
	Minimax	"Intermediate"	14
	Rinn	"Intermediate"	3
			25
2	*Medium-Speed Films*		
	DuPont	550 "D"	10
	Eastman Kodak	"Radiatized"	69
			79
6	*Fast Films*		
	I.F.I.	"Super-Speed"	7
	Minimax	"Extra-Fast"	62
	Rinn	"Extra-Fast"	0
			69
	Fastest Films		
9	Eastman Kodak	"Ultra-Speed"	22
12	DuPont	"Lightning-Fast"	5
			27

*Based on tests made by the Division or Oral Diagnosis, Tufts University School of Dental Medicine, and on information published by A. G. Richards (J. Am. Dent. A. 54: 476, 1957); and by A. H. Wuehrmann (ORAL SURG., ORAL MED. & ORAL PATH. 12: 1091, 1959).

and recent graduates, suggests a considerable block to encouragement of the use of high-speed films in conjunction with extended time-temperature processing, a procedure quite critical to their successful use. The limited information sought on the frequency of developer changes indicated that these were not made in relation to weekly work loads or any other well-based appraisal of factors contributing to developer exhaustion or deterioration.

TABLE IV. SAMPLE CHARACTERISTICS
DEVELOPING SOLUTIONS AND PROCESSING TECHNIQUES

BRAND OF DEVELOPER	TOTAL X-RAY UNITS SERVED	DEVELOPMENT BY INSPECTION	TIME-TEMPERATURE DEVELOPMENT (DATA ADJUSTED FOR 68° F.) 1-2 MIN.	3-4 MIN.	5-6 MIN.	7 MIN.	MEAN DEVELOPING TIME (MINUTES)
AA	24	13	2	6	2	1	3.7
DuPont	12	6	1	2	2	1	4.3
FR	66	33	6	24	3	0	3.3
G. E.	13	1	6	2	4	0	3.1
Kodak	79	38	9	28	4	0	3.3
Rinn	1	0	1	0	0	0	2.0
Other	2	2	1	0	0	0	1.0
	197	92	26	62	15	2	3.3
Films sent out for processing:	3						
Total	200						

PRIMARY RADIATION DOSAGE TO PATIENTS

In Table V the primary dosage accruing to patients exposed under the conditions described for our sample is reported in terms of roentgens in air at cone tip (skin surface), per film exposure and per complete intraoral x-ray

TABLE V. FACTORS IN PRIMARY DOSAGE TO PATIENTS
GENERAL FINDINGS

PRIMARY DOSE PER INTRAORAL FILM EXPOSURE		PRIMARY DOSE PER COMPLETE INTRAORAL X-RAY EXAMINATION	
ROENTGENS PER FILM MEASURED IN AIR AT SKIN SURFACE	NUMBER OF X-RAY MACHINES	ROENTGENS IN AIR AT SKIN SURFACE PER COMPLETE EXAMINATION	NUMBER OF X-RAY MACHINES
0.2- 0.4	10	Not over 5	10
0.5- 1.4	65	6- 15	35
1.5- 2.4	47	16- 25	46
2.5- 3.4	33	26- 35	26
3.5- 4.4	12	36- 45	32
4.5- 5.4	15	46- 55	14
5.5- 6.4	2	56- 65	8
6.5- 7.4	4	66- 75	9
7.5- 8.4	7	76- 85	4
8.5- 9.4	3	86- 95	3
9.5-10.4	1	96-105	5
10.5-11.4	0	106-115	2
11.5-12.4	0	116-125	1
12.5-13.4	0	126-135	3
13.5	1	136-145	0
	200	146-155	1
		156-165	1
			200
Mean: 2.64 r per film Median: 2.03 r per film Range: 0.15-13.5 r		Mean: 36.6 r per patient Median: 32.5 r per patient Range: 2.2-158 r	

examination. Mean values may appear disappointingly high to those who have hoped for and worked toward a substantial and general reduction in dosage, in the face of the extensive publicity accorded to radiation protection problems in both professional and lay media. However, while it may seem that unusual

TABLE VI. FACTORS IN PRIMARY X-RAY DOSAGE TO PATIENTS
DIMENSIONS OF THE X-RAY BEAMS

DIAMETER OF PRIMARY X-RAY BEAM AT SKIN SURFACE* (INCHES)	NUMBER OF X-RAY MACHINES	AREA OF SKIN SURFACE EXPOSED PER FILM† (SQUARE INCHES)
2.50	18	4.9
2.75	43	5.9
3.00	45	7.1
4.00	76	12.5
5.50	18	23.7
	200	
Mean: 3.51 Range: 1.5-6.0		Mean: 9.62 Range: 1.8-28.3

*Measured at cone tip and grouped according to most commonly encountered values for beam diameter.

†Compare area of standard size (#2) intraoral dental film—2.00 square inches.

efforts to educate dentists in the simple mechanics of dosage reduction have been relatively unproductive, it should be noted that the mean dose per film exposure (2.64 r) was only about one-half of an average dose quoted as recently as three years ago by the National Academy of Sciences[3] and reliable enough at that time. Unfortunately, it is still ten times as high as an average dose per exposure well established as adequate for the routine production of films of excellent diagnostic quality in a number of dental offices in this area and in the dental clinics of Tufts University. The situation invites the conclusion that more general action to minimize patient dosage, while possible through a revitalized concept of professional responsibility, will more likely stem from the widespread legislative activity now taking place.

The ensuing four tables offer convincing evidence in support of the effectiveness of simple procedures widely advocated for minimizing dosage to the patient. A conical x-ray beam confined to a diameter of 2.75 inches at cone tip will provide a generous minimum coverage of over 6.0 square inches at the film, whereas the standard-size dental film is exactly 2.0 square inches in area. From Table VI it can be deduced that 47.0 per cent of the machines were producing a beam whose area could well be reduced by one-half or more and still provide ample coverage for effective beam direction onto the film area. This would have the desirable result of sharply reducing the tissue area receiving x-rays as each film is exposed.

Radiation dosage depends in part on the number of films exposed, and this will rightly vary according to the diagnostic requirements of individual radiologists and the individual needs of their patients. In this respect, it is heartening to find evidence in Table VII that those operators who felt it necessary to base a complete intraoral x-ray examination on larger average numbers of exposures had apparently also taken effective steps to keep the associated dosage under control. It is not insignificant that those who used an average of from eighteen to twenty-two films per complete examination were involving their patients in a total primary dose that was virtually no higher than that given by the dentists who used between eight and twelve films for their examinations.

Practical proof of the effectiveness of increased filtration is offered by Table VIII, but 78.0 per cent of the x-ray machines were found to be operated with less than the 2.00 mm. aluminum total equivalent filtration recommended by the American Academy of Oral Roentgenology[5] and required by law in several states at this time. Added filtration, effective in its own right for reducing dosage, can also serve to supplement other devices (extended anode-to-film distance and lower milliamperage) for solving problems with mechanical exposure timers related to the use of faster films.

Both filtration and kilovoltage should be used to modify the half-value layer of the x-ray beam to produce the most efficient penetration of tissues for our diagnostic purposes. Of the two, filtration is the more efficient in reducing dosage, but higher kilovoltage (Table VIII) can also offer a significant contribution. The dose level for the 60 to 70 KVP range was generally

TABLE VII. FACTORS IN PRIMARY X-RAY DOSAGE TO PATIENTS
NUMBER OF EXPOSURES PER COMPLETE INTRAORAL X-RAY EXAMINATION

NUMBER OF FILMS USED FOR A COMPLETE INTRAORAL EXAMINATION (INCLUDING INTERPROXIMAL FILMS)	NUMBER OF X-RAY MACHINES	PRIMARY DOSE IN AIR AT CONE TIP			
		ROENTGENS PER FILM		ROENTGENS PER EXAMINATION	
		MEAN	RANGE	MEAN	RANGE
8 to 12 films (Mean—11.25)	35	2.98	0.33-13.5	37.2	4.0-148.5
14 to 16 films (Mean—14.92)	147	2.43	0.15- 9.6	37.5	2.4-134.4
18 to 22 films (Mean—18.66)	18	2.15	0.15- 7.9	38.8	2.2-158.0
	200				

TABLE VIII. FACTORS IN PRIMARY X-RAY DOSAGE TO PATIENTS
FILTRATION AND KILOVOLTAGE

	NUMBER OF X-RAY MACHINES	ROENTGENS IN AIR AT CONE TIP PER FILM EXPOSURE	
		MEAN	RANGE
Total Filtration			
Less than 1.00 mm. aluminum equivalent	83	3.89	1.00-13.50
1.00-1.75 mm. aluminum equivalent	73	1.97	0.40- 5.40
2.00 mm. and over, aluminum equivalent	44	1.23	0.15- 3.75
	200		
Operating Kilovoltage			
45-55 KVP	44	3.09	0.20-13.50
60-70 KVP	144	2.53	0.15- 9.00
90 KVP	5	0.47	0.20- 0.70
Undetermined KVP	7		
	200		

TABLE IX. FACTORS IN PRIMARY X-RAY DOSAGE TO PATIENTS
FILM EMULSION SPEEDS AND DEVELOPING TIMES

	NUMBER OF X-RAY MACHINES	ROENTGENS IN AIR AT CONE TIP PER FILM EXPOSURE	
		MEAN	RANGE
*Film Emulsion Speed**			
1—Slow	25	4.48	1.25-13.50
2—Medium	79	3.49	0.75- 9.60
6—Fast	69	1.59	0.20- 8.00
9, 12—Fastest	27	1.36	0.15- 2.00
	200		
Film Developing			
Time-Temperature at 68° F.			
2 minutes or less	26	3.10	0.44- 8.80
3-4 minutes	62	2.50	0.15- 8.40
5 minutes or more	17	3.23	0.15- 9.60
	105		
Development by inspection	92	2.62	0.30-13.50
Sent out for processing	3	1.88	0.70- 2.70
	200		

*See Table III.

18.1 per cent lower than that prevailing for the 45 to 55 KVP range. Hasty conclusions should not be drawn from the data for 90 KVP machines, as their number is insufficient to provide a statistically significant basis.

We have indicated that filtration is a major instrument for the reduction of x-ray dosage; the utilization of higher film speeds can prove even more dramatically effective (Table IX). Use of the correct exposure times for full development of films of varying emulsion speeds will, if other factors are held equal, produce a dosage change inversely proportional to their relative emulsion speeds. Since the proportional relationship is not literally evidenced in Table IX, it would appear that full advantage had not been taken of the dosage reduction potential inherent in the use of fast films. Either unnecessarily long exposure times were being incurred in the case of fast films or less attention was being paid to other dosage-reduction factors when these films were used.

Table IX also relates dosages to processing procedures. For our sample there was no demonstrated association between longer processing times and lower primary doses—nor, for that matter, between dosages related to time-temperature film processing and to development by visual inspection. Due to the phrasing of one of the questions used, it is possible that in a number of instances reported as development by inspection there was also partial reliance on a fixed development time factor, but this explanation could not affect the import of the findings under discussion. We venture to suggest that because the relationship between dosage per exposure and film-processing time under well-controlled conditions has been clearly and repeatedly established, the whole procedure generally involved in the development of films in dental offices may be thrown into some disrepute by the findings in Table IX. It seems probable that in dental offices no practical results will emerge from emphasis on extended processing times until and unless the customary processing facilities and techniques are considerably upgraded.

SCATTER RADIATION TO OPERATORS

Under the conditions studied, prevailing levels of scatter x-ray dosage to operators are reported in Table X. There has been comparatively less public concern for the potential occupational hazard in dental roentgenography, but there were certainly some instances in our sample in which unacceptable levels were disclosed in respect of whole-body radiation dosage. It is true that 34.5 per cent of the operators were receiving no measurable scatter radiation, and, for all practical purposes, the 22.0 per cent reported as receiving marginal doses (no higher than 5 mr per 100 film exposures) may be added to bring this total to a more welcome 56.5 per cent. However, 32.5 per cent of the operating locations involved scatter radiation doses of from 6 to 54 mr per 100 films, and another 6.0 per cent of the operators were exposed to scatter radiation exceeding this level (in one case this was as high as 168 mr per 100 films). As has been stated already, it is a simple matter to reduce the primary dosage received by

TABLE X. FACTORS IN SCATTER RADIATION TO OPERATORS
GENERAL FINDINGS

SCATTER DOSAGE PER 100 FILMS*		SCATTER DOSAGE PER WEEKLY WORK LOAD*		
MILLIROENTGENS IN AIR PER 100 FILM EXPOSURES	NUMBER OF X-RAY MACHINES	REPORTED WEEKLY AVERAGE NUMBER OF EXPOSURES	NUMBER OF X-RAY MACHINES	MILLIROENTGENS IN AIR PER WEEK (MEAN AND RANGE)
0	69†	*Operator NOT Shielded from Patient's Head:*		
1- 5	44	5- 25 films	44	4.25 (0-25.2)
6- 15	36	30- 50 films	65	6.49 (0-41.5)
16- 25	17	55-100 films	33	9.67 (0-58.8)
26- 35	10	105-300 films	27	21.80 (0-115.0)
36- 45	8		169	
46- 55	4			
56- 65	1			
66- 75	5	*Operator Behind Door, Partition, or Lead Screen:*	31	0
76- 85	2	Mean: 63 films		
86- 95	1	Range: 15-125		
96-105	1		200	
Over 105:				
150	1			
168	1			
	200			
Mean: 13.75 mr		Mean: 7.59 mr		
Median: 3.50 mr		Median: 2.10 mr		
Range: 0-168		Range: 0-115		

*Measured approximately 42 inches above floor level at operating location during intra-oral exposure with film positioned behind upper central incisor teeth.

†Including thirty-one machines operated from behind door, partition, or leaded screen.

TABLE XI. FACTORS IN SCATTER RADIATION TO OPERATORS
CHARACTERISTICS OF THE PRIMARY BEAM

	NUMBER OF X-RAY UNITS (N = 169)*	MILLIROENTGENS IN AIR PER 100 FILM EXPOSURES†	
		MEAN	RANGE
Beam Diameter at Cone Tip			
2.50 inches or less	13	2.6	0-12
2.75 inches	40	7.0	0-82
3.00 inches	40	13.5	0-92
4.00 inches	60	19.7	0-168
5.50 inches	16	47.1	0-75
Operating Kilovoltage			
45-55 KVP	39	14.5	0-150
60-70 KVP	120	17.2	0-168
90 KVP	3	3.0	1-4
Undetermined kilovoltage	7		
Primary Beam Total Filtration			
Less than 1 mm. aluminum equivalent	75	22.6	0-168
1.00-1.75 mm. aluminum equivalent	62	10.5	0-90
2.00 mm. and over aluminum equivalent	32	12.4	0-66
X-ray Output at Cone Tip			
0.2- 1.5 r per exposure	61	5.50	0-27
1.5- 2.4 r per exposure	41	15.20	0-92
2.5- 5.4 r per exposure	49	20.00	0-82
5.5-13.5 r per exposure	18	42.80	0-168

*Excluding thirty-one units operated from behind a screen or partition (see Table XII).

†Measured at operating location, approximately 42 inches above floor, during upper incisor exposures.

the patient to minimal levels; it should be emphasized at this point that it is even simpler for the dentist to eliminate all scatter radiation for himself or, as the case may be, for his hygienist or assistant.

All the factors which contribute to primary dosage—beam area, half-value layer, and amount of radiation—affect the amount of scatter radiation emitted from the subject exposed to the x-ray beam, some to a greater extent than others. In Table XI the beam area or diameter was found in practice to have a dramatic effect on scatter radiation, this factor being associated with strikingly low mean levels when held to a diameter of 2.75 inches at cone tip (31.4 per cent of the machines). Likewise, the lower outputs per film (not over 1.5 r) had sharply reduced associated scatter doses, evidenced in 36.1 per cent of the instances. The relationship between scatter dosage and factors determining the half-value layer of the beam (filtration and kilovoltage) is more complex because of their influence on beam penetration characteristics. Considered together, as they should be for this purpose, when increased or raised they do contribute somewhat to a reduction in scatter radiation, but not to an extent paralleling the effectiveness of other factors. This is evident from the relevant findings in Table XI.

TABLE XII. FACTORS IN SCATTER X-RAY DOSAGE TO OPERATORS
DISTANCE FROM SOURCE OF SCATTER RADIATION

LOCATION OF OPERATOR (DISTANCE FROM HEAD OF PATIENT)	NUMBER OF MACHINES	MILLIROENTGENS IN AIR PER 100 FILM EXPOSURES*	
		MEAN	RANGE
Distance if no intervening screen or partition:			
2 feet or less	11	48.4	7-168
3 feet	28	33.7	0-150
4 feet	52	14.3	0- 97
5 feet	35	10.1	0- 67
6 feet	27	6.1	0- 42
7 feet or more	16	2.0	0- 12
	169	16.3	0-168
Operator behind screen or intervening partition:			
(Regardless of distance)	31	0	0
	200		

*Measured at approximately 42 inches above floor, at location of operator, during upper incisor exposures.

Table XII indicates that almost any kind of partition or screen will eliminate all scatter radiation to the operator. (Fully leaded screens or adequate barriers are necessary, however, for the protection of adjacent areas from the primary beam.) The distance of an unscreened operator was found to be effective, as could be expected, almost in inverse proportion to its squared value. Increasing distance, however, while effecting a striking reduction in scatter dosage, is by itself no guarantee of radiation safety when the upper limits of the ranges in Table XII are taken into account. This point is raised to counter an impression created by constant emphasis on distance as a precautionary measure. Distance will serve well to achieve this objective only when accompanied by low primary dosages from well-collimated x-ray beams.

TABLE XIII. SUMMARY OF GENERAL FINDINGS FOR X-RAY DOSAGE IN DENTAL OFFICES

FINDINGS	PER CENT OF INSTALLATIONS
Primary Dose per Film Exposure	
Less than 0.5 r per film	5.0
0.5-1.4 r per film	32.5
1.5-3.4 r per film	40.0
3.5-5.4 r per film	13.5
5.5 r or over	9.0
Primary Dose per Complete Intraoral X-ray Examination	
Not over 5 r per examination	5.0
6- 15 r per examination	17.5
16- 35 r per examination	36.0
36- 55 r per examination	23.0
56-100 r per examination	12.0
Over 100 r per examination	6.5
Scatter Radiation to Operator per 100 Film Exposures	
No measurable scatter radiation	35.0
1- 5 mr per 100 films	22.0
6- 25 mr per 100 films	26.0
26- 50 mr per 100 films	11.0
51-100 mr per 100 films	5.0
Over 100 mr per 100 films	1.0
Scatter Radiation to Operator per Weekly Work Load	
No measurable scatter radiation	35.0
1- 5 mr per week	34.0
6- 25 mr per week	23.0
26- 50 mr per week	5.0
51-100 mr per week	2.5
Over 100 mr per week	0.5

TABLE XIV. COMPARISON OF HIGH-DOSAGE AND LOW-DOSAGE TECHNIQUES (MEANS AND RANGES OF EXTREME DECILES OF RANK ACCORDING TO PRIMARY DOSAGE PER FILM EXPOSURE)

	LOWEST 10 PER CENT		HIGHEST 10 PER CENT	
*Primary Dosage**				
Roentgens per film exposure	0.45	(0.15-0.60)	7.77	(5.40-13.50)
Roentgens per complete examination	6.5	(2.2-10.0)	102.4	(48.5-158.0)
Technical Factors:				
Total filtration (mm. aluminum equivalent)	1.89	(0.75-2.54)	0.64	(0.50-1.13)
Operating kilovoltage (KVP)	66.5	(45-90)	58.8	(45-65)
Relative film speed†	6.75	(6-12)	1.63	(1-2)
Films per complete examination	15.1	(10-22)	14.2	(10-20)
Developing time (minutes at 68° F.)	3.7	(2-7.5)	4.4	(1-5)
Scatter Radiation‡				
Milliroentgens per 100 exposures	3.0	(0-7)	51.0	(0-168)
Milliroentgens per weekly work load	0.81	(0-6)	12.4	(0-59)
Technical Factors:				
Beam diameter (inches at cone tip)	3.08	(2.12-6.00)	3.94	(2.75-5.50)
Primary dose per film (r at skin)§	0.45	(0.15-0.60)	7.77	(5.40-13.50)
Distance of operator from patient (feet)	4.1	(2.5-7.0)	3.4	(1.5-6.0)

*Roentgens in air at skin surface.

†See Table III for explanation.

‡Milliroentgens in air measured approximately 42 inches above floor level at operating location during intraoral exposure with film positioned behind upper central incisors.

§See section above ("Primary Dosage") for primary dose factors.

RECAPITULATION

Table XIII recapitulates the general findings for levels of primary dosage to patients and scatter radiation to operators in terms of the percentages of installations operated at various dosage levels. Table XIV further emphasizes the findings reported for individual factors in dosage, through study of the exposure factors characteristic of the highest and lowest deciles of primary dosage per film exposure. Comparing the operation of x-ray machines involved in the highest and lowest deciles, we find that for the former group both primary dosage to the patient and scatter dosage to the operator were more than seventeen times as high as in the lowest 10 per cent. The desirable results achieved by the latter group are seen to be largely associated with the use of films that are four times as fast, total filtration triple that of the other group, beam areas that are less by more than one-third, and operating distances (squared) 50 per cent greater. The comparison demonstrates quite vividly what could be generally achieved in actual practice by an informed profession alert to its responsibility for the protection of patients and occupationally exposed personnel.

DISCUSSION

It should be useful to repeat this type of study from time to time in order to observe trends in prevailing levels of radiation dosage to patients and to operators and the extent to which radiation protection measures are adopted. Experience suggests the incorporation of certain features to enhance the value of future survey programs. These may be indicated briefly as follows:

1. Determination of the half-value layer of each x-ray beam (in terms of aluminum thickness).
2. Duplicate film exposures at each installation, using a standard step-wedge and the dentist's average exposure factors and films—one film to be processed in the dentist's customary manner and the other centrally processed under well-controlled conditions.
3. "Spinning-top" film checks for the accuracy of each exposure-timing device, at standard intervals over its likely range of utilization.
4. Objective data for determining the exposure per work load for each installation (separately for each operator).
5. Development of a durable manikin on which to make standardized test exposures at each installation, valid for evaluating the diagnostic acceptability of films and for the measurement of scatter radiation.
6. Authoritative and reliable data concerning various errors inherent in survey measurements from ionization chamber instruments under field conditions (that is, in private dental offices).[2]
7. Reliable density curves for dosimeter films (if used) well related to the range of half-value layers represented by the various x-ray beams being studied.[1]

CONCLUSIONS AND SUMMARY

Mean dose rates were well within the maximum permissible limits established by the National Committee on Radiation Protection and were found to be only about one-half as great as dosages reported three years previously. Mean dose rates, however, remained relatively high in comparison with levels being attained routinely through the use of simple measures for minimizing dosage. Findings in this survey fully support the effectiveness of these measures under dental office conditions. Periodic surveys of mean radiation dosage levels are desirable to permit continuing evaluation of the progress toward optimum standards of radiation protection in dental offices.

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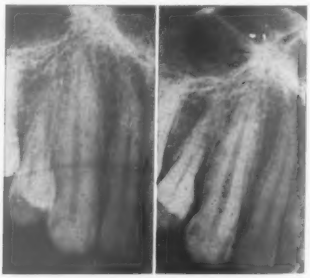
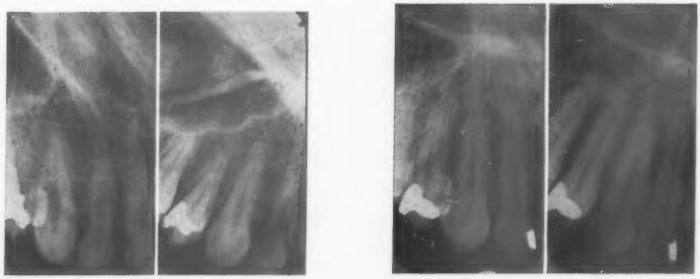
HORIZONTAL ANGULATION IN MAXILLARY CUSPID ROENTGENOGRAPHY

Robert E. Silha, B.S., D.D.S., M.S., Iowa City, Iowa*

MAXILLARY cuspid roentgenograms are probably poorly angulated more consistently than are roentgenograms of any other area of the mouth. The most common cause of a poor diagnostic roentgenogram in this area is an overlapping of the contact point on the distal aspect of the cuspid. Usually such overlapping is exaggerated by the prominent lingual cusp of the maxillary first premolar (Fig. 1).



A. *B.*



A. *B.* *A.* *B.*

Fig. 1.—Roentgenograms showing incorrect *(A)* and correct *(B)* horizontal angulations in the same mouth.

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It has been observed in the Oral Diagnosis Clinic of the State University of Iowa College of Dentistry that the distal contact of the cuspid usually can be seen with no overlapping on bitewing films (Fig. 2). It has also been noted that the labial surface of the upper cuspid lies in the same plane as the buccal surfaces of the posterior teeth rather than in the plane of the labial surfaces of the central and lateral incisors (Fig. 3). These two observations suggest a solution to maxillary premolar-cuspid overlapping.

The common tendency is for the operator to position the cone so that the beam from the x-ray tube enters the arch too much from the anterior. In other words, in thinking of the cuspid as the cornerstone of the arch, one assumes that the horizontal angulations used for the central incisors and the premolars should be bisected and the central ray for the cuspid directed along the bisection line. This tendency leads to the production of a poor roentgenogram, since the beam of x-rays will cut across the lingual cusp of the first premolar. The result is an overlapping of this cusp onto the distal contact point of the cuspid (Fig. 4).

Fig. 2.—Premolar and bitewing roentgenograms of the same patients as those shown in Fig. 1.

When the cone is directed from a more distal aspect, the beam will go through the contact points of the cuspid with no overlapping. About the same horizontal angulation should be used for the cuspid as for the premolars. However, the cone (the useful beam of radiation) must be moved far enough forward to cover the film with radiation (Fig. 5).

SUMMARY

A better diagnostic roentgenogram of the upper cuspid will result if correct horizontal angulation is used. The prominent lingual cusp of the maxillary first premolar can be prevented from superimposing onto the distal aspect of the cuspid if the operator will direct the x-rays through the premolar-cuspid interproximal space.

Fig. 3.

Fig. 4.

Fig. 5.

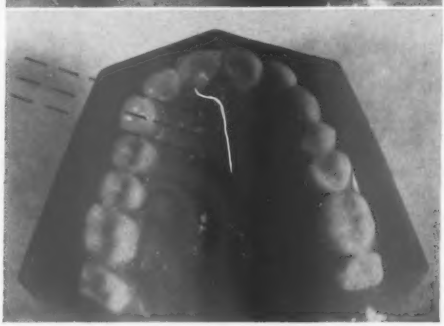


Fig. 3.—Model of patient's mouth showing relationship of the labial surface of the cuspid to the labial and buccal planes of the incisors and premolars, respectively.

Fig. 4.—Model showing bisection of incisor and premolar horizontal angulations and the x-ray beam cutting across the lingual cusp of the first premolar.

Fig. 5.—Model showing the x-ray beam going through the contacts of the cuspid when the correct horizontal angulation is used.

ROENTGENO-QUESTIONS

Why does there often appear to be a rarefaction about the apices of roots superimposed over the maxillary sinus, mandibular canal, or mental foramen?

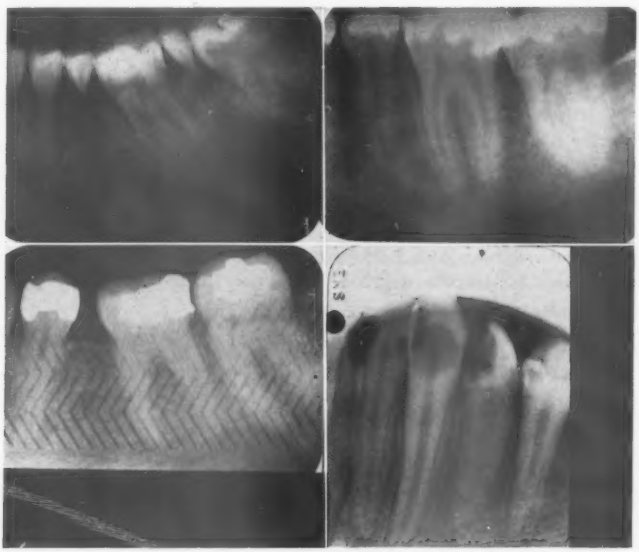
Density or darkness of an area on a dental x-ray film is determined, among other things, by the amount of radiation absorbed in the tissues as the radiation passes through such structures. The more radiation absorbed, the lighter will be the area on the film; the more radiation transmitted, the darker will be the area. Thus, when radiation passes through a body cavity, such as a maxillary sinus, a foramen, or a canal, there is less radiation absorbed than in the surrounding calcified tissues. Since radiation passes through such cavities, there results an area of greater density on the film; this is in contrast with the immediately surrounding tissues. The superimposition of the root apex on such a roentgenolucent area often gives the illusion of a thickened periodontal space. Apparent thickening of the space, especially when correlated with extensive caries, can be erroneously interpreted as evidence of apical pathology. An understanding of such rarefactions is important if misinterpretation is to be avoided. If a second film is taken, with a shorter exposure, all surrounding areas may appear too light on the film. The dense areas questioned will be more interpretable, however, and one can then visualize more effectively the periodontal membrane space, the lamina dura, etc.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

Technical Errors

Although the films below are not oddities in the true sense since they occur with fairly considerable frequency, I thought that the readers of the "Roentgo-Oddities" section might be interested in identifying several rather consistently observed, yet possibly puzzling, errors. The upper left-hand film demonstrates a double exposure. The upper right-hand one shows movement of the film in relation to the teeth during exposure; the lower left-hand film illustrates a



Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

herringbone pattern superimposed over the teeth and is indicative of film exposure through the backing rather than through the exposure side of the film (all metal film backings do not have such a herringbone pattern impregnated into them and it is therefore possible to expose a film through the incorrect surface without producing this herringbone pattern); and the lower right-hand film shows a typical appearance of cone cutting. Cone cutting has become more frequent since emphasis has been placed on the need for columnizing with lead diaphragms in an effort to reduce irradiation. Such cone cutting is not necessary if the operator is careful in his angulating techniques.

Author's name withheld by request.

ORAL PATHOLOGY

BENIGN NEOPLASTIC FIBRO-OSSEOUS LESION OF THE MANDIBLE

Report of a Case

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INTRODUCTION

SEVERAL authors believe that such fibro-osseous lesions as ossifying fibroma or fibro-osteoma are variants of fibrous dysplasia and under no circumstances represent an independent neoplastic disease entity.[1-5] The present case is reported to illustrate the pertinent features which provide ample evidence that there may exist, as has already been pointed out by Thoma,[6] Waldron,[7] and others,[8] a benign neoplastic disease similar in histologic structure to fibrous dysplasia of bone.

CASE REPORT

A 26-year-old white woman was referred to the Division of Oral Surgery on Nov. 7, 1956, by the Obstetrics Department of the University Hospital for evaluation of a unilateral facial swelling. Since the patient had been married for six years but had no children, her physician had sent her to the hospital for gynecologic examination. The physician had also noticed the facial swelling and had suggested its investigation. The patient's recollection concerning the duration of the swelling was vague. She stated that she had been aware of it for several years but had noticed its rapid increase in size over the last two years. The swelling had caused her no discomfort, and therefore she had not sought any medical advice. She had a past history of chickenpox, measles, mumps, and possibly rheumatic fever at the age of 12 years. Her family history was noncontributory.

Physical examination revealed a well-developed, well-nourished young woman who was in no acute distress but had an apparent facial asymmetry due to a swelling of her right cheek (Fig. 1). The swelling extended from the tragus of the right ear to the submandibular region and anteriorly to the nasolabial fold. The swelling was diffuse, firm in consistency, and nonflammatory. The overlying skin was freely movable and of normal texture and color. Pertinent intraoral findings were limited to the right mandible. Expansion of

From the Divisions of Oral Pathology (A. P. C. and R. J. G.) and Oral Surgery (R. J. B. and A. R. S.), School of Dentistry, University of Minnesota.

the buccal cortical plate, extending from the lateral incisor posteriorly to include the ascending ramus, was noted. There was minimal distortion of the lingual plate of bone. Mobility of the second premolar was demonstrated, and the second and third molars were missing although there was no history of tooth removal in this area.

Extraoral roentgenograms revealed a large, expansive osteolytic lesion occupying the major portion of the body and ascending ramus of the right mandible (Figs. 2 and 3). Throughout the lesion there was a suggestion of the presence of foci of calcification. An unerupted tooth was also seen within the mass. Unlike a malignant neoplasm, the lesion had regular borders. On the basis of long-standing history and clinical and roentgenographic examination, a tentative diagnosis of (1) massive dentigerous cyst, (2) ameloblastoma, and (3) fibrous dysplasia was considered. On Nov. 9, 1956, several biopsy specimens were obtained via an intraoral approach. Microscopic study revealed that the tissue was composed of fibroblasts arranged in whorls with a very small amount of intervening fibrous connective tissue. Foci of calcification and atypical bone spicules were present throughout the specimen. The histologic picture was typical of fibrous dysplasia (Fig. 4).

Fig. 1.—Photograph showing facial asymmetry due to a swelling of the right cheek.

The patient was admitted to the hospital for surgical removal of the tumor. Laboratory studies, such as urinalysis, differential blood count, bleeding and clotting time, serum calcium and phosphorus, and alkaline phosphatase, were within normal limits. On Nov. 11, 1956, under general anesthesia, the right posterior teeth were extracted and access to the tumor mass was gained by elevation of a large mucoperiosteal flap and removal of a section of buccal cortical bone. The marrow space was found to be replaced by tissue that was friable, hemorrhagic, and meaty in consistency. Attempts at removal of the tumor resulted in profuse hemorrhage which was controlled with great difficulty. The operation was stopped short of complete removal of the tumor mass. Blood loss was replaced by 1,000 c.c. of whole blood and 1,000 c.c. of 5 per cent dextrose in water.

Microscopic examination of the tissue removed at operation showed features similar to those seen in the previous biopsy. The postoperative course was uneventful, and the patient was discharged with instructions to return in two weeks for reappraisal of her condition. Subsequently, she developed an abscess of the cheek which was incised and drained intraorally.

On Jan. 8, 1957, she was readmitted to the University Hospital for a second attempt at complete surgical removal of the tumor. Following ligation of the external carotid artery, the tumor mass was completely removed through an intraoral approach, and the defect was packed loosely with iodoform gauze. Microscopic study of the excised tissue substantiated the diagnosis of fibrous dysplasia. Recovery was uneventful, and the patient left the hospital one week after the operation. The iodoform dressings were maintained by her family physician for one month.

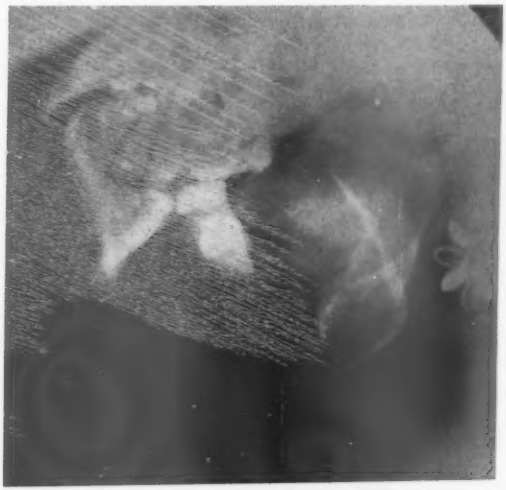

Fig. 2.—Lateral jaw roentgenogram showing a large unilocular, osteolytic lesion involving the body and ascending ramus of the mandible. Note small foci of calcification and an unerupted tooth present in the roentgenolucent area.

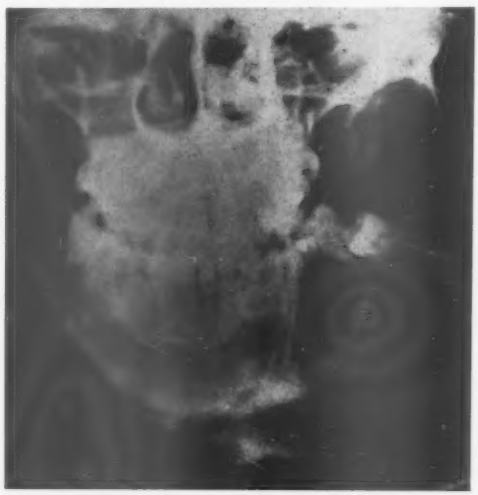

Fig. 3.—Posteroanterior roentgenogram showing the extent of the destructive lesion. Note the marked bulging of the buccal plate of bone.

The patient was again seen on Feb. 19, 1957, at which time the operative site showed the presence of hyperplastic tissue along the margins of the bony defect, extending back to the anterior faucial pillars. Clinically, it appeared to be a keloidlike response of the oral mucosa. However, biopsy of the tissue indicated recurrence of the tumor in the deeper

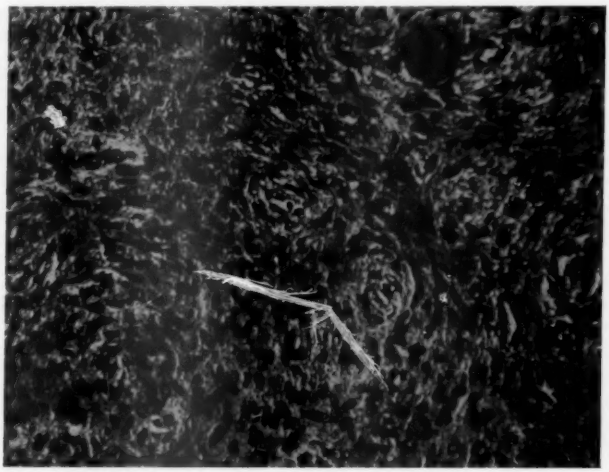

Fig. 4.—Photomicrograph of tissue from the first biopsy. The specimen is composed chiefly of fibroblasts arranged in whorls with scant intercellular connective tissue. Note a focus of calcification in the top corner.

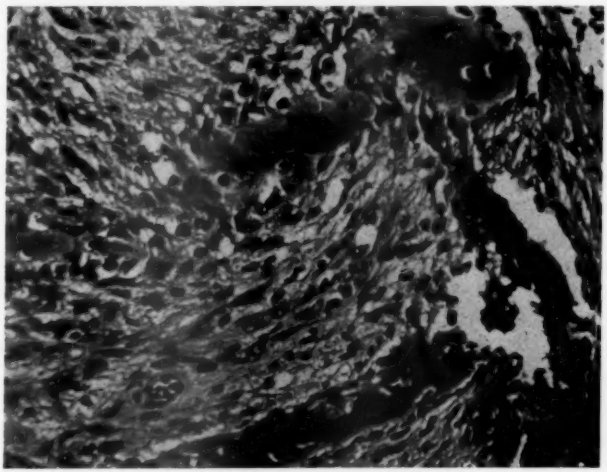

Fig. 5.—Photomicrograph of hyperplastic tissue removed from around the bony margins. There is marked fibroblastic and osteoblastic activity in this specimen. Note the bone spicules surrounded by osteoid tissue and covered with a layer of osteoblasts.

portions. The tumor, which exhibited less cellularity than was seen in the previous sections, was composed of whorls of connective tissue interspersed with atypical bone spicules (Fig. 5). Under local anesthesia, the hyperplastic tissue was completely excised.

One month later, recurrence was again evident about the wound margins as well as within the bony defect. The patient was referred to the department of surgery for further management. On March 25, 1957, the recurrent tumor was removed via an intraoral approach. In order to minimize facial asymmetry, an attempt was made to collapse the buccal

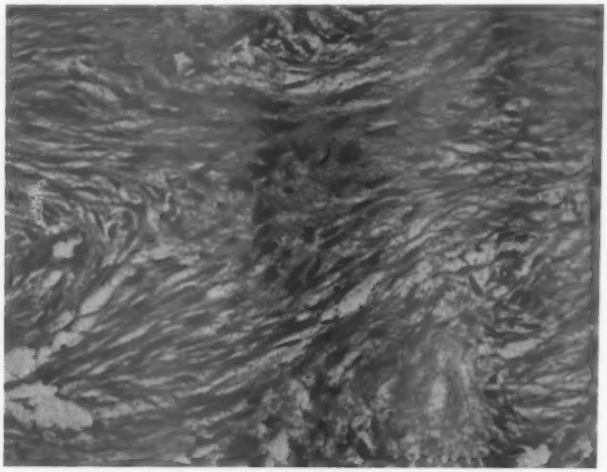

Fig. 6.—Photomicrograph of specimen obtained from the resected mandible. Note that the tissue is chiefly made up of fibrous tissue, with few fibroblasts.

Fig. 7.—Photograph showing postoperative results following hemimandibulectomy. The vertical scar resulted from ligation of the external carotid artery.

cortical plate. This, however, resulted in fracture of the mandible, and intermaxillary fixation became necessary. The patient was discharged from the hospital one week after surgery. Intermaxillary fixation appliances were removed by a local dentist three weeks postoperatively.

On June 15, 1957, the patient returned to the hospital. Her right cheek was swollen and red, and she was unable to open her mouth. Hot packs and antibiotics rendered the patient asymptomatic and afebrile. Intraoral examination revealed recurrence of the lesion within the depth of the bony cavity. On July 1, 1957, following a prophylactic tracheotomy, a hemiresection of the right mandible below the neck of the condyle was performed. Gross and microscopic examination of the resected mandible showed recurrent tumor. The microscopic appearance was characterized by an increase in the fibrous element over that noted in the previous sections (Fig. 6). When the patient was last seen on Jan. 22, 1959, there was no evidence of the tumor's recurrence.

DISCUSSION

The term "ossifying fibroma" was possibly initiated by Montgomery[9] in 1927 to designate a neoplasm composed of fibrous tissue interspersed with varying amounts of newly formed bone. According to Sherman and Sternberg,[8] the neoplasm was first described in 1872 by Menzel, who considered it to be a form of osteoma. In 1939 Eden,[10] from a study of five cases, concluded that "ossifying fibroma" represents an immature form of the benign fibro-osseous tumors of the membranous bone. According to Thoma,[6] ossifying fibroma or fibro-osteoma, terms used for the neoplastic fibro-osseous lesions, represents a "cellular fibroma, in which, according to the stage of differentiation attained, various amounts of a variety of calcified intercellular substance are produced."

Lichtenstein and Jaffe,[1] in 1942, coined the term "fibrous dysplasia of bone" to include fibro-osseous lesions which were known in the literature by a variety of names but had more or less similar histologic features. These lesions are believed to arise from faulty embryogenesis. Jaffe[2] further suggested that the lesions previously diagnosed as ossifying fibroma were not neoplastic in nature but were variants of fibrous dysplasia of bone. Since then, the existence of ossifying fibroma as a neoplastic disease has been denied by several other investigators.[1-5] It is possible that in the past several cases of fibrous dysplasia were erroneously diagnosed as neoplastic fibro-osseous lesions, but it seems also true that at present cases of the latter diseases are being confused with fibrous dysplasia. It does not seem justifiable that the two distinct disease entities with different pathogeneses should be considered as one simply because they manifest themselves in similar microscopic features. The roentgenographic appearance and clinical course of neoplastic fibro-osseous lesions are sufficiently diagnostic to permit differentiation of neoplastic fibro-osseous lesions from fibrous dysplasia of bone.

Fibrous dysplasia of bone develops in early life, and its greatest growth occurs before puberty. Due to the lack of symptoms, however, the lesion may not be discovered until late in life. After puberty, fibrous dysplasia either becomes static or its increase in size becomes imperceptible. Ossifying fibroma, on the other hand, may occur at any age and is progressive in growth. The patient in the case just described noticed a rapid increase in size of the swelling at an age when fibrous dysplasia usually becomes quiescent. Roentgenographically, the lesion in question was unilocular—an important finding pointed out by Sherman and Sternberg[8] and Waldron[7] in their descriptions of cases

of neoplastic ossifying fibroma. Rarely, if ever, does fibrous dysplasia cause such extensive destruction of bone as was evident in this patient. Furthermore, loosening of teeth is seldom demonstrable in fibrous dysplasia, but it is not an uncommon finding in neoplastic disease. Fibrous dysplasia causes expansion and distortion of bone in all directions, unlike neoplastic fibro-osseous lesions in which the displacement is usually in one direction. There was hardly any noticeable expansion of the lingual plate of bone in our patient. In fibrous dysplasia the involvement of bone is diffuse, and there is no line of demarcation between the lesion and the surrounding bone. On the other hand, the neoplastic ossifying fibroma may not be encapsulated but there is a distinct boundary between the tumor mass and the bone, as was seen in the present case. Fibrous dysplasia is usually avascular. Its vascularity is rarely so marked as to demand ligation of the external carotid artery, but Montgomery has recommended that such surgical procedures be performed in cases of extensive lesions of ossifying fibroma because of the marked vascularity of these lesions. Furthermore, repeated recurrence of the lesion at intervals of less than four weeks and its extension into the surrounding soft tissues are more in keeping with the behavior of a neoplastic disease than of fibrous dysplasia of bone.

SUMMARY

We have described a case in which neoplastic fibro-osseous lesion occurred in a 26-year-old white woman. We have also discussed pertinent features which may permit differential diagnosis of the neoplastic ossifying fibroma from fibrous dysplasia of bone. The roentgenographic appearance and clinical course of fibrous dysplasia and neoplastic ossifying fibroma are sufficiently diagnostic to permit their differentiation from one another.

The authors are grateful to Dr. H. B. Clark, Jr., Chairman of the Division of Oral Surgery, with whose permission this case has been reported.

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ENDODONTICS

The American Association of Endodontists

Captain Warren J. Hedman, Editor

IDIOPATHIC INTERNAL RESORPTION OF TEETH

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ALTHOUGH idiopathic internal resorption is comparatively rare and its etiology is unknown, correction by endodontic therapy has been achieved. Two cases successfully treated will be reported here.

CASE 1.—A 26-year-old Negro man presented a pink discoloration on the crown of the upper central incisor. The tooth was asymptomatic and noncarious. Its response to the electric pulp tester was within the normal range, and it was not sensitive to percussion. Clinical examination revealed that the tooth had a pink color when viewed from the labial side. On the lingual side the pink color was more noticeable. There was a minute perforation through the lingual enamel, and the underlying pulp tissue was exposed. A roentgenogram revealed that the pulp canal was of normal size in the root portion, but a very wide roentgenolucent area could be observed in the crown of the tooth.

There was no history of trauma to this tooth. Blood differential counts and blood chemistry studies revealed normal values (see Fig. 1).

CASE 2.—An 18-year-old Negro girl was seen for oral rehabilitation and examination. She had poor oral hygiene, chronic gingivitis, and extensive carious lesions involving the pulps of several teeth. Roentgenographic examination of the tooth in question revealed no abnormality of the pulp chamber or pulp canal at this time. An inlay was present in the crown of the tooth, and there had been no recurrence of caries. Six months later, however, a new set of roentgenograms revealed an oval roentgenolucent lesion in the pulp canal midway between the apex and the crown of this tooth. The tooth was comfortable and responded normally to the electric pulp tester. The tooth was not sensitive to percussion. The inlay was removed, and under the inlay there was no recurrence of caries. A wall of clinically sound dentine separated the floor of the cavity preparation from the pulp chamber. There was no history of trauma to this tooth, and all blood studies were normal (see Fig. 3).

DISCUSSION

Internal resorption is usually an asymptomatic process which may extend over a period varying from a few months to several years. It is seen most commonly in the upper anterior teeth, although any tooth of the deciduous or

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permanent dentition may be affected.[1] The process may involve the coronal or the radicular portions of the tooth. If the coronal area is involved, a pink discoloration of the crown is evident, and the roentgenogram will show a wide roentgenolucent area in the crown. Mummery[2, 3] described such a tooth, and it has since been known as the "pink tooth of Mummery."

Fig. 1. Fig. 2.

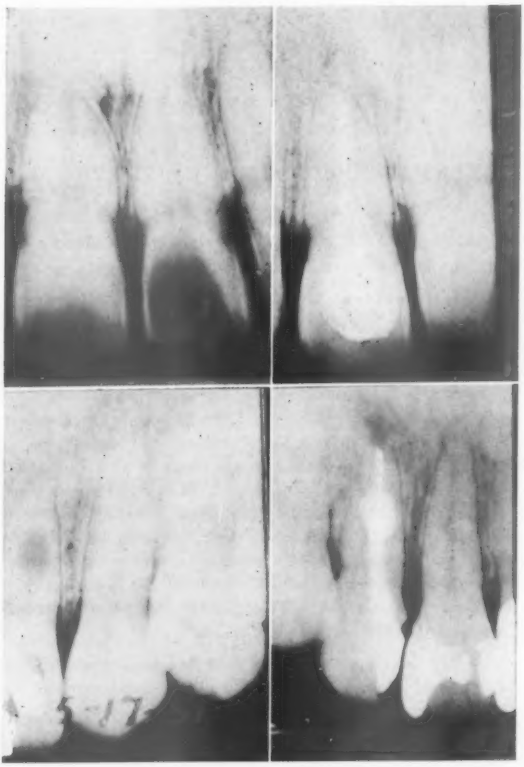

Fig. 3. Fig. 4.

Fig. 1.—Roentgenolucent area observed in crown of central incisor.
Fig. 2.—Roentgenogram taken after endodontic treatment, showing root canal and roentgenolucent area filled.
Fig. 3.—Oral roentgenolucent area in pulp canal.
Fig. 4.—Roentgenogram taken after endodontic treatment, showing root canal and roentgenolucent area filled.

If the root portion is involved, there may be no clinical evidence of the lesion's existence but the condition will show in the roentgenogram. Resorption in the root may result without discomfort to the patient and may lead to a perforation of the root if the pulp is permitted to remain in the tooth. The process has been known to perforate through the dentine, cementum, and enamel of the tooth, and fracture of the tooth has occurred after extensive resorption of the root dentine.

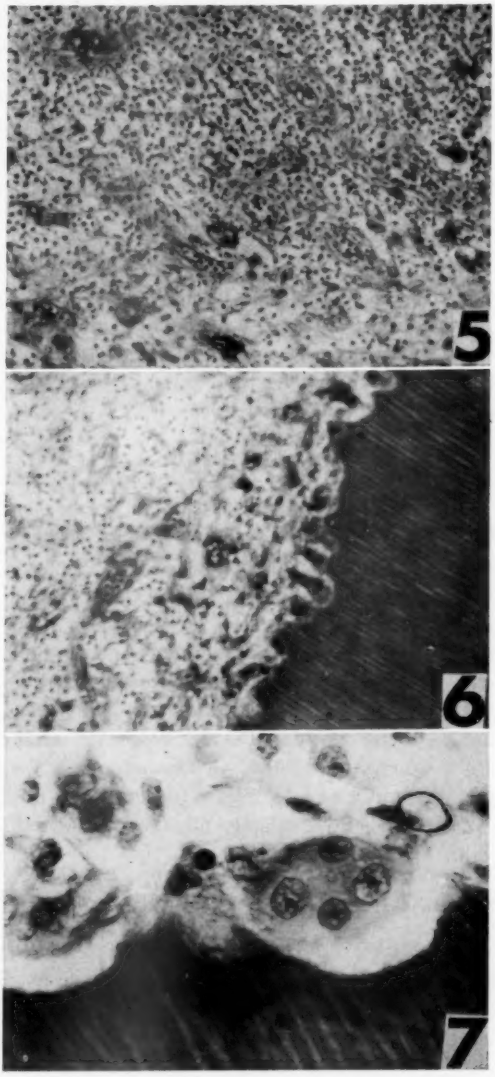

Fig. 5.—The pulp tissue resembles granulation tissue and consists of proliferating fibroblasts, numerous thin-walled blood vessels, and a profuse infiltration of lymphocytes or plasma cells. (Magnification, ×264; reduced ⅓.)

Fig. 6.—The odontoblasts have disappeared and the pulpal surface of the dentine is lined with multinucleated giant cells. (Magnification, ×550; reduced ⅓.)

Fig. 7.—Giant cells (osteoclasts) in baylike excavations are observed in the dentine. (Magnification, ×1164; reduced ⅓.)

HISTOPATHOLOGY

In cases of idiopathic internal resorption the pulp tissue consists of a well-vascularized granulation tissue instead of the usual delicate connective tissue bordered by odontoblasts. Numerous lymphocytes and plasma cells are found throughout the tissue in the pulp chamber and pulp canal. The odontoblasts have disappeared in the area of resorption, and in their places along the dentine wall are found multinucleated giant cells. Baylike excavations into the dentine are present, indicating active resorption of the dentine. These are similar to the baylike excavations observed when osteoclasts resorb bone. Thoma[4] suggests that these cells be called odontoclasts, since dentine and not bone is being resorbed here. He suggests that the lesion be called odontoclastoma. (See Figs. 5, 6, and 7.) The resorptive activity may continue through the dentine, cementum, or enamel, resulting finally in the perforation of the root or the crown of the tooth. In some cases metaplasia of the pulp has been observed, and bone formation has occurred in this lesion.[5-10] In such cases osteoblasts will be found associated with the newly formed bone.

TREATMENT

Treatment of this condition must be either removal of the pulp tissue followed by endodontic therapy or extraction of the tooth. The latter may be necessary in the event of root perforation.

In the two cases cited here, it was fortunate that through roentgenographic examination and clinical observation the lesions were discovered early. The pulp was extirpated in each case and after proper endodontic therapy the canals were filled. Some difficulty was encountered in the filling of the root canal, especially in the bulbous resorbed areas. This was achieved by first flooding the canal with the root-canal-sealer cement, and then, using a gutta-percha cone with a pumping motion, the cement was forced laterally and apically. Finally, the gutta-percha cone remnant was pushed into the root canal, and the canal was then completely filled with additional pieces of gutta-percha (Figs. 2 and 4).

SUMMARY

Two cases of idiopathic internal resorption have been presented, and the treatment of this disorder has been discussed. These cases illustrate the value of periodic roentgenographic examination and careful clinical observation. The involved teeth were saved for these patients by early diagnosis and endodontic treatment.

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STEREOMICROSCOPIC STUDY OF 700 ROOT APICES OF MAXILLARY AND MANDIBULAR POSTERIOR TEETH

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THIS study is a supplement to my previous article entitled "A Stereomicroscopic Study of the Root apices of 400 Maxillary and Mandibular Anterior Teeth."[1] I have thus completed the analysis of posterior as well as anterior teeth.[2]

The purpose of this article is to establish with greater precision than has heretofore been attained the morphology of the canal at the root apex, the major apical foramina, and accessory apical foramina.

In examining past and current literature, I noted that in previous investigations of pulp canal morphology the techniques employed in the preparation of the teeth for study may have distorted the specimens. Therefore, any descriptions, evaluations, or conclusions drawn from such studies may have been inaccurate.

The use of the stereomicroscope has given a view of these areas in depth and has provided a better opportunity to observe, analyze, and compare anatomic areas.

In this study, the basic principle was to use specimens which had been carefully treated and prepared so as not to mar the natural detail. The specimen apices were cut at a 7 mm. distance from the apex and cemented, flat end down, on glass microscope slides. The apices were then studied under the three-dimensional microscope, which had a micron measuring device on the lens. The measurements of the foraminal openings, as noted in Tables I and II, were tabulated to two decimals.

Considerable judgment had to be used in classifying round, oval, irregular, and funnel-shaped openings, as many of these shapes were of the borderline variety. Some orifices which appeared to be foramina, such as indented cementum, seemed to enter the main canal, but gradations of hard to soft cementum and dentine made classification difficult.

In order to determine accurately the percentage of accessory foramina present, it was also necessary to explore, penetrate, and study the cut ends of

This study was exhibited at the International Centennial of the American Dental Association in New York, New York, Sept. 14-18, 1959; the meeting of the American Association of Endodontists in Chicago, Illinois, February, 1958; and the midwinter meeting of the Chicago Dental Society, February, 1959, and February, 1960.

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TABLE I. DESCRIPTION OF MAJOR APICAL FORAMINA OF MAXILLARY AND MANDIBULAR POSTERIOR TEETH

TEETH	NUMBER OF ROOTS EXAMINED	AVERAGE DIAMETER OF MAJOR APICAL FORAMINA EXAMINED (MM.)	AVERAGE DISTANCE FROM APEX (MM.)	MAJOR FORAMINA (PER CENT)					
				DIRECTLY ON APEX	ROUND CONTOUR	OVAL CONTOUR	ASYMMETRICAL CONTOUR	SERRATED PERIPHERY	PRONOUNCED FUNNEL
*Maxillary molars**									
Lingual root	100	0.4	0.5	31	53	29	18	21	15
Distobuccal root	100	0.4	0.45	27	46	39	15	3	34
Mesiobuccal root	100	0.35	0.4	40	55	29	16	9	27
Mandibular molars									
Mesial root	100	0.5	0.45	34	52	35	13	24	14
Distal root	100	0.65	0.45	47	42	47	11	21	19
Maxillary									
First premolars	50	0.3	0.3	47	71	18	11	7	34
Second premolars	50	0.3	0.4	46	58	19	23	9	27
Mandibular									
First premolars	50	0.35	0.5	36	66	22	11	6	40
Second premolars	50	0.3	0.45	60	55	24	20	13	20

*All molar calculations include first and second molars.

all roots. A Kerr No. 1 file was used to explore the cut end, as well as the apex, for major and accessory foramina. This instrument measured about 1 micron (0.001 mm.) at its tip and flared to a thickness of 125 microns at a point 1 mm. from the tip. Canal orifices at the cut end were from approximately 0 to 1 mm. or more in diameter.

In entering a canal from either the cut end or the apical end, negotiation was simple, effective, and successful in about 90 per cent of the cases. In the

TABLE II. DESCRIPTION OF ACCESSORY APICAL FORAMINA OF MAXILLARY AND MANDIBULAR POSTERIOR TEETH

TEETH	NUMBER OF TEETH EXAMINED	AVERAGE DIAMETER (MM.)	AVERAGE DISTANCE FROM APEX (MM.)	ACCESSORY FORAMINA (PER CENT)			
				TOTAL WITH ACCESSORY FORAMINA	ONE FORAMEN	TWO FORAMINA	THREE OR MORE FORAMINA
*Maxillary molars**							
Lingual root	100	0.25	1.0	13	11	2	None
Distobuccal root	100	0.2	0.85	17	12	4	1
Mesiobuccal root	100	0.2	1.0	32	20	10	2
Mandibular molars							
Mesial root	100	0.2	0.8	18	10	3	5
Distal root	100	0.3	0.8	35	23	5	7
Maxillary							
First premolars	50	0.15	1.0	34	29	3	2
Second premolars	50	0.15	1.0	32	15	10	7
Mandibular							
First premolars	50	0.2	1.0	45	19	15	11
Second premolars	50	0.15	1.0	47	25	17	5

*All molar calculations include first and second molars.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

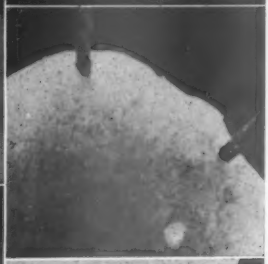


Fig. 5.

Fig. 6.

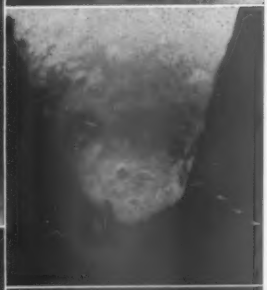
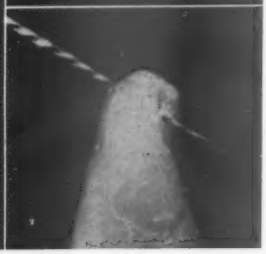


Fig. 7.

Fig. 8.



Figs. 1 to 9.—Photomicrographs showing unusual variations in morphology of apical foramina. A Kerr No. 1 file was used for exploration.

Fig. 1.—Maxillary molar, mesiobuccal root. The lingual canal foramen is on the right. On the left are two foramina, possibly a major and accessory or else the result of a bifurcated major canal.

Fig. 2.—Mandibular first premolar. A No. 1 file is inserted through a lateral canal which follows a course at a right angle to the major canal.

Fig. 3.—Roots of a maxillary first premolar. Note incompletely developed apical foramina.

Fig. 4.—Apex of a maxillary second premolar. Note major apical foramen at 45 degree angle to axis of root and 2 mm. short of apex. An accessory foramen appears below.

Fig. 5.—Maxillary first premolar. Canals are exiting in unparallel manner.

Fig. 6.—Mandibular first premolar with multiple foramina.

Fig. 7.—Palatal root of maxillary second molar with one major foramen and two accessory foramina.

Fig. 8.—Maxillary first premolar. A canal follows a horizontal course 2 mm. below the apex. The major canal meets the horizontal canal.

remaining 10 per cent the following difficulties were encountered: (1) Varying degrees of calcified material in the canal prevented successful negotiation of or even entrance into the canal from either end. (2) In a marked curve in a canal a thin, flexible instrument would follow through. In some cases a broader, more rigid instrument would not follow the curve but instead would cause a ledge, after which negotiation of the apex, even with the finer instrument, was not possible. (3) A bent instrument had no propelling power.

To supplement the data of this study, I am now preparing a paper entitled "A Study of Double Canals in Single Roots," the results of which will alter somewhat the percentages of major and accessory foramina. Since it was not expedient to cut the 7 mm. root apices used in this study for sagittal examination, a new set of 1,100 teeth was classified. In cutting roots sagittally, I found that many had two canals at the 7 mm. level which coalesced to form a single canal between this point and the apex. In other cases a major canal bifurcated to two canals about 2 to 3 mm. short of the apex. In rare cases these, in turn, bifurcated again, making four or five multiple foramina.

Fig. 9.—Maxillary second premolar. Note dens in dente.

The mesiobuccal roots of the upper molars presented a noteworthy aspect. In twenty-eight cases of apparent double canals at the cut end, the analysis was as follows:

1. Twelve had two major canals at the cut end as well as at the apex. One specimen had two roots (similar to a premolar).
2. In sixteen cases one of the two major canals was calcified and impenetrable. Of these, thirteen had impenetrable lingual canals, and three had impenetrable buccal canals.

Impenetrable canals were found less often in other roots having double canals.

In classifying single roots with possibilities of double canals, I found that some had one major canal and that some had two. This group included premolars, mesiobuccal roots of upper molars, and mesial roots of lower molars. Where there was only one major canal, it was untenable to classify it as a buccal or lingual canal. Descriptive analyses of buccal and lingual canals were found to be quite similar. Because of these findings, buccal, lingual, and single canals were averaged under one heading.

In Tables I and II it is to be noted that two-rooted premolars were included in the averages, but in order to avoid confusion they were not stated in the tables.

In Table I, total percentages add up to more than 100, since foramina had more than one classification. Accessory foramina were, for the most part, round in shape and classification was not indicated.

In some cases it was not possible to detect with anatomic accuracy the designation of a first and second molar; hence, their inclusion under one heading.

Approximately 25 per cent of the maxillary first premolars and about 10 per cent of the maxillary second premolars have two fully formed roots. In the mandibular premolars the occurrence of two roots is a rare anomaly.

SUMMARY

1. By comparison with previous methods, studying the specimens in their original and natural conditions and shapes may have resulted in more accurate findings.

2. Average accessory foramina are about one-half the size of major foramina and twice the distance from the apex.

3. Approximately 50 per cent of major foramina open directly on the apex. Those which do not open at the apex range from eccentric position to 2 mm. from the apex.

4. The average funnel in an apical foramen diminishes in diameter to about one-half its size at a point about 0.75 mm. from the surface opening.

5. While Tables I and II are nearly accurate for application in about 90 per cent of cases found in the oral cavity, allowances should be made for variables and imponderables.

6. When there was a curve at the root end, the canal invariably followed the curve rather than a straight line.

7. In multirooted teeth, whether accessory foramina were or were not present, the other roots usually had similar conditions.

CONCLUSION

I have endeavored to add to the present knowledge of apical morphology by organizing my observations into definite statistical form. It is hoped that this analysis will stimulate further investigations in this field of study.

Appreciation is hereby expressed to Dr. Arnold Reisman of the Watson Laboratories of The International Business Machine Company for his assistance in the investigation and calculation of averages, as well as to The International Business Machine Company for permission to use their equipment.

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RESEARCH

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RESPONSE OF THE PERIODONTIUM, PULP, AND SALIVARY GLANDS TO GINGIVAL AND TOOTH INJURY IN YOUNG ADULT MALE RATS

II. Pulp and Periapical Tissues

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THE report on the first part of this study included a description of the materials and methods used.[1] The present investigation deals with the response of pulpal and periapical tissues to experimental injury involving mechanical exposure of the pulp.

OBSERVATIONS

For the sake of clarity, the changes noted in the tissues will be described by time intervals.

Immediately After Injury (Fig. 1).—Exposure of the pulp had occurred. An increase of cells near the injury was noted. The vessels of the pulp were prominent and appeared to be full of cells. No periapical changes were seen. Fuchsinophilia was not observed at the site of pulpal injury.

Three Hours After Injury (Fig. 2).—The pulp became increasingly cellular, with many cells aggregating near the site of injury. The pulpal vessels were markedly prominent. No periapical changes were observed. A slight fuchsinophilia was noted in the fibers near the injury.

Six Hours After Injury.—The pulpal contents appeared largely necrotic. In areas of viable pulp tissue, attempts at secondary dentine formation were observed (Fig. 3). An increase in the vascularity and cellularity of the periapical connective tissue was noted. The pulpal tissue below the injury showed marked fuchsinophilia, which decreased after hyaluronidase digestion. Beta metachromasia and a slight increase in argyrophilic fibers were also noted in

From the Murry and Leonie Guggenheim Foundation, Institute for Dental Research, and the Department of Periodontia and Oral Medicine, New York University College of Dentistry.

This study was supported in part by Grant-in-Aid #D-537 from the Institute for Dental Research, National Institutes of Health, United States Public Health Service, Bethesda, Maryland.

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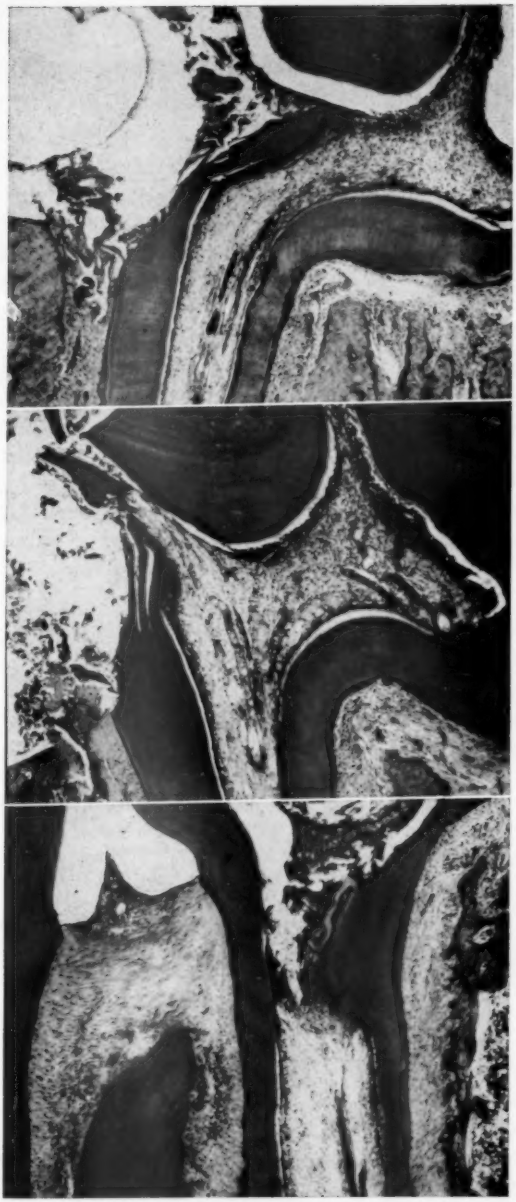


Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1.—Photomicrograph of a mesiodistal section of the maxillary first molar of a rat sacrificed immediately after injury. (Hematoxylin and eosin stain. Magnification, ×60; reduced 3/10.)

Fig. 2.—Photomicrograph of a mesiodistal section of the maxillary first molar of a rat sacrificed three hours after injury. (Hematoxylin and eosin stain. Magnification, ×80; reduced 3/10.)

Fig. 3.—Photomicrograph of a mesiodistal section of the maxillary first molar of a rat sacrificed six hours after injury. (Hematoxylin and eosin stain. Magnification, ×80; reduced 3/10.)

the area. The periapical tissue showed an increase in argyrophilic and fuchsinophilic fibers, which also stained beta metachromatic (Fig. 4).

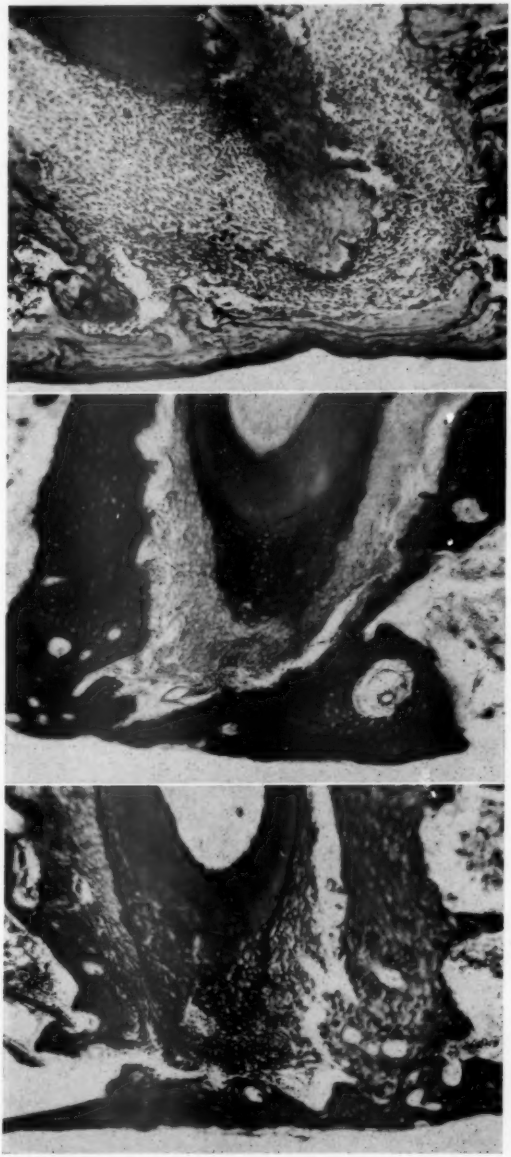
One to Three Days After Injury (Fig. 5).—Many polymorphonuclear leukocytes were seen near the area of injury. Aggregates of necrotic pulpal tissue below injury were also noted, as was edema throughout the pulp. After twenty-four hours, polymorphonuclear leukocytes and an increasing number of capillaries were observed in the periapical area. These changes became more evident with an increase in time. Pulpal tissue below the injury stained markedly fuchsinophilic, as did the periapical fibers. An increase in argyrophilic fibers was also noted around the apex.

Four to Six Days After Injury (Fig. 6).—The pulp exhibited a large necrotic mass below the injury, surrounded by a dense accumulation of inflammatory cells. The periapical tissue showed an inflammatory cellular infiltrate, an increase in vascularity, and many young disoriented fibroblasts. The alveolar bone surrounding the apex was undergoing active resorption. Fuchsinophilic fibers were present, surrounding the necrotic pulpal tissue and the apex. An increase in argyrophilic fibers was noted around the necrotic pulpal mass but was more evident in the tissue surrounding the apex.

Seven to Fourteen Days After Injury (Fig. 7).—Pulpal tissue showed increasing necrosis, becoming completely necrotic by eight to nine days. The periapical tissues showed an increasing amount of granulation tissue, which reached the maximum extent at about ten days. By fourteen days after injury, the granulation tissue was slowly being replaced by organizing connective tissue which also appeared to wall off the inflammatory cell elements at the apical foramen. The alveolar bone surrounding the apex showed evidence of osteoclasis and osteogenesis in different areas. The fibers around the apex showed fuchsinophilia, which was markedly decreased after hyaluronidase digestion. Metachromasia was not noted. Silver impregnation showed reticulin fibers among increasing collagen around the apex.

Fifteen to Twenty-two Days After Injury.—No marked changes from those described in the previous period were noted. The pulps were completely necrotic, and periapical healing as evidenced by better organization of the periapical tissue and increased osteogenesis around the apex appeared to be prevalent in many specimens. Reticulin fibers were gradually being replaced by collagen fibers in this area.

Twenty-three to Forty-four Days After Injury.—The inflammatory infiltrate around the apical foramen appeared well localized. The periapical tissue seemed to be largely composed of oriented collagen bundles. Osteogenesis was observed at the alveolar bone surface surrounding the apex. It should be pointed out, however, that resorption of the periapical bone was initially quite extensive, at times involving the interradicular septum. While osteogenesis was noted at the bone surfaces at this time, the repair activity never replaced the extensive amount of bone resorbed initially. It should also be noted that the degree of healing present varied within the specimens of the group. In the



A.

B.

C.

Fig. 4.—*A*, Photomicrograph of apical tissues of the distal root of the maxillary first molar of a rat sacrificed six hours after injury. (Hematoxylin and eosin stain. Magnification, ×100; reduced 3/10.)

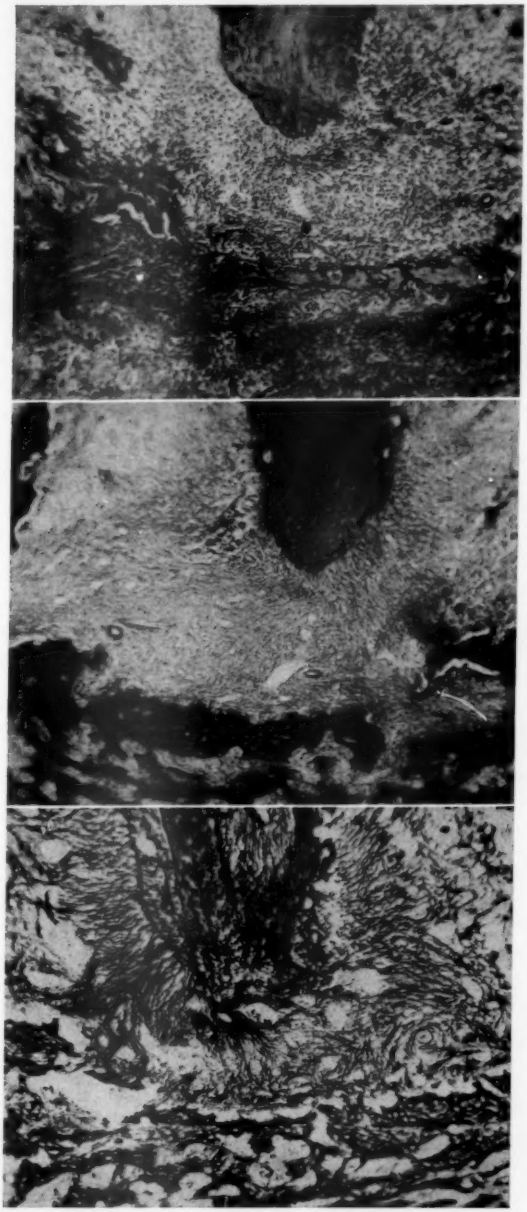
B, Serial section of specimen shown in *A*. (P.A.S. stain. Magnification, ×100; reduced 3/10.)

C, Serial section of specimen shown in *A*. (Silver impregnation stain. Magnification, ×100; reduced 3/10.)

Fig. 5.—*A*, Photomicrograph of apical tissues of the distal root of the maxillary first molar of a rat sacrificed forty-eight hours after injury. (Hematoxylin and eosin stain. Magnification, ×100; reduced 3/10.)

B, Serial section of specimen shown in *A*. (P.A.S. stain. Magnification, ×100; reduced 3/10.)

C, Serial section of specimen shown in *A*. (Silver impregnation stain. Magnification, ×100; reduced 3/10.)



A.

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Fig. 6.—*A*, Photomicrograph of apical tissues of the distal root of the maxillary first molar of a rat sacrificed six days after injury. (Hematoxylin and eosin stain. Magnification, ×100; reduced 3/10.)

B, Serial section of specimen shown in *A*. (P.A.S. stain. Magnification, ×100; reduced 3/10.)

C, Serial section of specimen shown in *A*. (Silver impregnation stain. Magnification, ×100; reduced 3/10.)

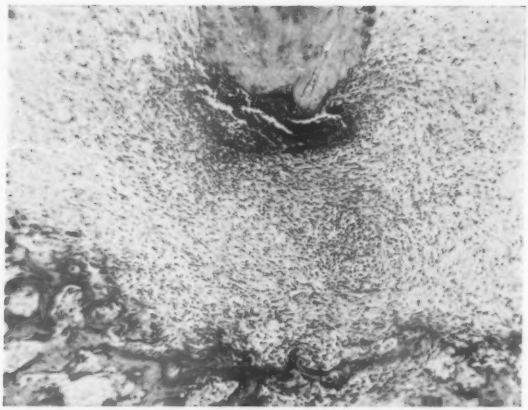
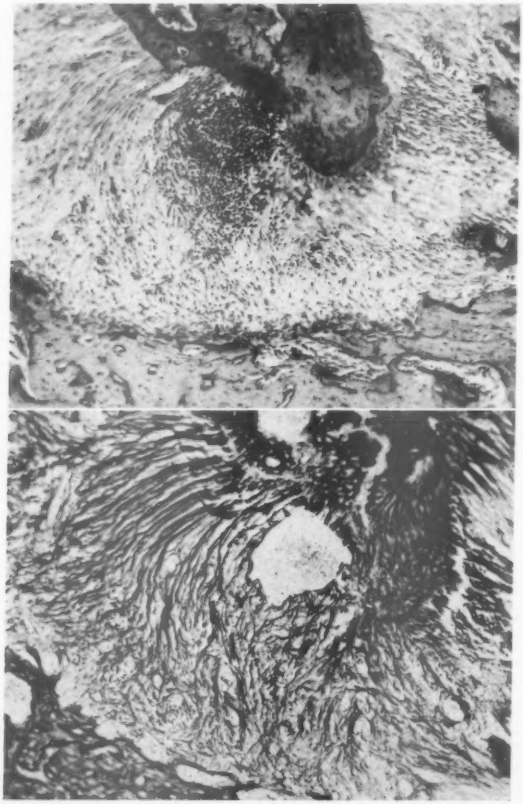


Fig. 7.—Photomicrograph of apical tissues of the distal root of the maxillary first molar of a rat sacrificed fourteen days after injury. (Hematoxylin and eosin stain. Magnification, ×100; reduced 3/10.)

A.



B.

Fig. 8.—*A*, Photomicrograph of apical tissues of the distal root of the maxillary first molar of a rat sacrificed thirty-seven days after injury. (Hematoxylin and eosin stain. Magnification, ×100; reduced 3/10.)

B, Serial section of specimen shown in *A*. (Silver impregnation stain. Magnification, ×100; reduced 3/10.)

animals showing increasing organization of the periapical tissues, a marked reduction in fuchsinophilic and argyrophilic fibers was noted (Fig. 8).

Of special interest was the appearance of cysts at the apex, which first were noted thirty days after injury. While epithelial proliferation at the apex was not seen prior to thirty days after injury, it was observed in five out of twenty specimens thirty-plus days after injury (Fig. 9).

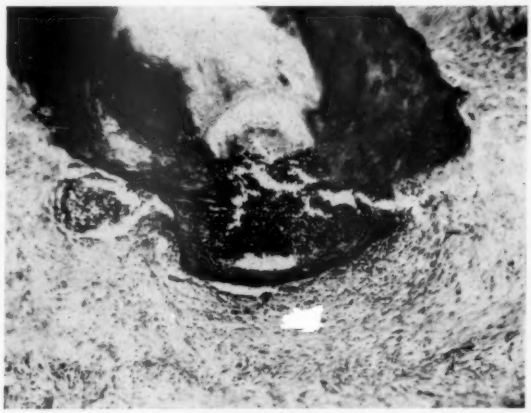


Fig. 9.—Photomicrograph of apical tissues of the distal root of the maxillary first molar of a rat sacrificed thirty-six days after injury. (Hematoxylin and eosin stain. Magnification, ×100; reduced 3/10.)

COMMENT

A comparison between gingival and pulpal tissue responses to experimentally induced injury showed differences which probably are the results of the different morphologic and anatomic configurations of the two types of tissue.[2] In my specimens pulpal exposure invariably led to complete necrosis of the pulp, while gingival injury led to gingival healing at a more apical level. Periapical tissue, on the other hand, appeared to organize more rapidly and showed more extensive collagen formation than did gingival tissue during the same time period. The reduced healing potential of the pulp might be explained on the basis of its anatomic position,[3] while the increased healing rate of the periapical tissues may be due to the relatively more protected position of this tissue as compared to the surface exposed gingiva. The behavior of these apical tissues is similar to that originally described by Blayney[4] and demonstrates the effects of different environments on wound healing.[5]

Histochemical observations of these tissues again showed an increase in fuchsinophilia with early stages of wound healing, followed by a reduction in this reaction when repair activity became more prominent. Further identification of this fuchsinophilic material showed it to be partially hyaluronidase labile and ortho to beta metachromatic, which was interpreted as being indicative of the presence of hyaluronic acid and mucoproteins.

Of interest was the relatively late appearance at the apex of epithelial proliferations which attempted to enclose the inflamed periapical tissue. This epithelium was not noted in specimens obtained less than 30 days after injury, but it was frequently seen in the older specimens. Since this epithelium is derived from the rests of Malassez and responds to inflammatory stimulus by proliferation,[6] the appearance of epitheliated dental granulomas thirty days after injury may indicate the time required for stimulation of this epithelium by inflammation.

SUMMARY AND CONCLUSIONS

Exposure of the pulp in young adult male rats led to pulpal necrosis within eight to nine days after injury. Inflammatory cells were noted within the periapical tissues twenty-four hours after pulpal injury. The inflammatory cellular infiltrate seen periapically increased in intensity, reaching its maximum four to six days following injury, after which time granulation tissue became increasingly evident. By fourteen days after injury, granulation tissue appeared to be gradually replaced by collagen fibers. This process continued and showed increasing organization of the collagen fibers with increasing time. Some specimens taken thirty-plus days after injury exhibited epithelial-lined dental granulomas.

Fuchsinophilia and argyrophilia were evident in the periapical fibers in the early stages following injury but decreased gradually as collagen formation increased.

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ORAL ANATOMY AND HISTOLOGY

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THE SIGNIFICANCE OF THE INNERVATION OF THE PREDENTINE

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INVESTIGATORS of the innervation of the teeth have recently reported that predentine contains more nerve fibrils in older persons than in younger persons.[1, 5] In order to study the nerve distribution of the predentine zone at advanced ages, which here is called secondary predentine, a number of teeth from persons aged 50 to 70 years were examined.

METHOD

The freshly extracted teeth were fixed in Bouin's solution, demineralized in 5 per cent nitric acid or 24 per cent EDTA, embedded in paraffin, cut in 7 μ serial sections, and stained according to the method of Palmgren,[4] which was described in detail in an earlier article.[1]

RESULTS

The predentine was found to contain many nerve fibrils, which coursed across the predentine zone in loops and curves between the pulpal and the dentinal borders. They generally made a fairly small angle with the tubules (Figs. 1 and 2). Where mineralization had begun, no nerve fibrils could be traced.

The thickness of the fibrils varied considerably, fine ones alternating with coarse ones (Figs. 1 and 3). Many fibrils were seen to widen suddenly into claviform or spindle-shaped structures (Figs. 3 and 4). Others were beaded (Figs. 1, 3, 5, and 6). Sometimes there was profuse ramification (Fig. 5). Occasionally the fibrils passed straight across the predentine zone (Figs. 6 and 7). Loops and curves were frequent (Figs. 1, 2, 7, and 8).

DISCUSSION

From reviews of the literature,[1] it is clear that there are three main conflicting opinions concerning the innervation of the teeth. While many research

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workers report finding nerve fibrils in the mineralized dentine, others claim that these fibrils penetrate no further than to the odontoblastema. Third, there are those who maintain that the fibrils pass into the predentine but not into the mineralized dentine.

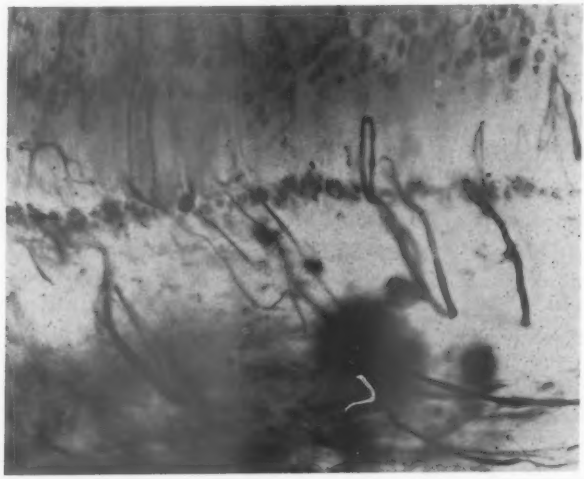

Fig. 1.—Nerve fibrils entering the predentine zone, where they form loops.

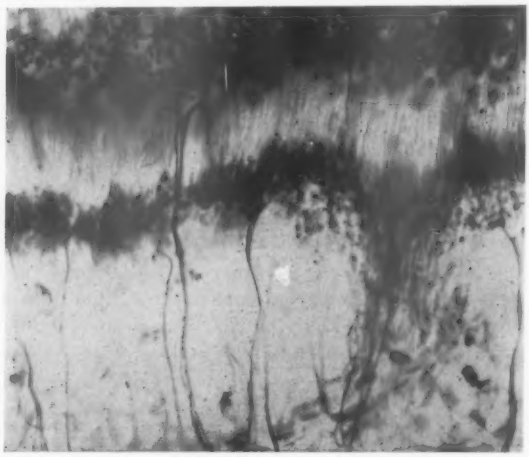

Fig. 2.—Nerve fibril entering the predentine zone, where it splits into finer fibrils, one of which proceeds to the dentinal junction while the others return toward the pulp.

It has been shown that in the earlier stages of the tooth's development no nerve fibrils exist in the vicinity of the dentinal matrix or the odontoblastema.[1] They seem to grow into the tooth papilla and to follow the capillaries. In teeth

from younger persons the nerve fibers seem to end in the odontoblastema or in the vicinity of the pulpopredentinal border membrane. An occasional single nerve fibril can be traced into the predentine, where it courses at an angle to

Fig. 3.

Fig. 4.

Fig. 3.—Nerve fibrils entering the predentine zone. One of the fibrils shows a coarse, clublike structure, probably due to damage and subsequent regeneration.

Fig. 4.—Nerve fibrils of the pulp. One of the fibrils enters the predentine zone and becomes a claviform structure suggestive of regeneration.

Fig. 5.—Nerve fibrils ramifying in the pulp. All branches enter the predentine zone, where some of them are split into finer fibrils. Thickening indicates damage to the fibrils and possibly regenerative changes.

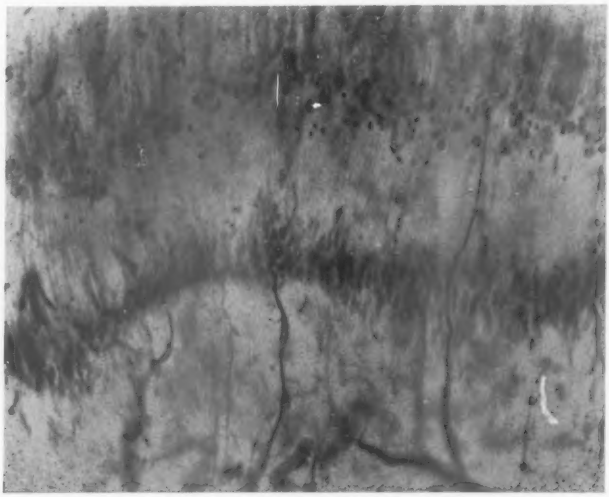

Fig. 6.—Nerve fibril crossing the predentine zone on a rather straight course but at a fine angle to the dentinal tubules. The structure resembling a nerve ending (middle right) may be due to damage of the fibril and subsequent regeneration.

the tubules.[1] In the predentine zone of adult teeth and in the corresponding zone of secondary dentine of the teeth of elderly persons, the nerve fibrils seem to exist in considerable numbers.

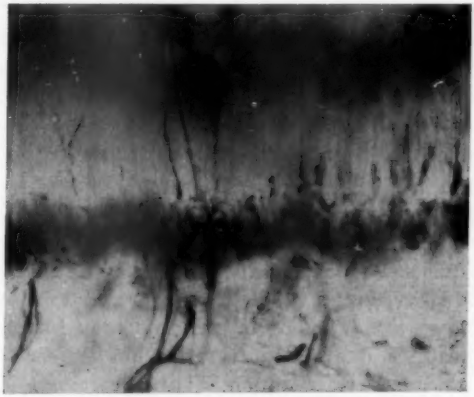

Fig. 7.—Nerve fibril crossing the predentine zone at a fine angle to the dentinal tubules and returning from the dentine-predentine junction.

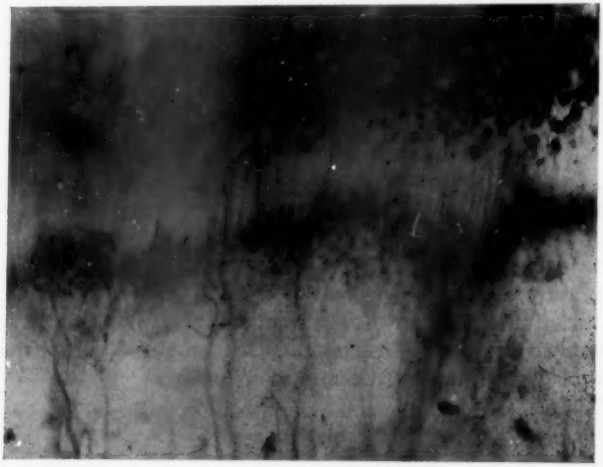

Fig. 8.—Nerve fibril curving in a wide arc back to the odontoblastema.

From this it may be concluded that the nerve fibrils issue from the central bundles of the pulp in a peripheral direction until their progress is halted by the already formed dentine. Teeth at this stage of development present the picture of noninnervated predentine. The nerve fibrils end in the odontoblastema or close to the pulpopredentinal junction. With the progressive formation of predentine, the nerve fibrils turn in loops and curves in the odontoblastic zone, and occasionally one will be enclosed in the newly formed predentine, as explained by Bernick.[2] This is often the case in younger persons.

In older persons, in whom the formation of secondary dentine is sometimes rather rapid and pronounced because of pathologic conditions, a large number of nerve fibrils are enclosed in the secondary predentine.[1, 5,7] It is also possible that delicate nerve fibrils, owing to their inherent growing ability, may be able to grow into empty spaces in the dentine itself, as is held by some investigators.[3]

It might be possible to explain the variety of pictures reported, and thus the divergent opinions on the innervation of the teeth, if only the ages of subjects were considered. Unfortunately, this is but rarely done. Moreover, the material has too often been limited to one tooth or perhaps to a few teeth at the most, which of course does not justify the drawing of general conclusions.

As to the significance of the nerve fibrils in the secondary predentine, their functional ability as pain conductors remains to be investigated. Envelopment by the secondary predentine is likely to impair the nerve fibril. In fact, the morphologic picture is analogous to that presented by damaged and regenerating nerves reported in other tissues.[6] The claviform and spindle-shaped swellings of the nerve fibrils, which have so often been interpreted as nerve endings, may thus be not only headings due to disintegration but also swellings of the fibrils caused by degeneration or regeneration. Through mineralization of the predentine, the enclosed nerve fibrils seem to decompose and thus may lose their function.

It is possible that in rare cases remnants of these degenerated or degenerating nerve fibrils in the mineralized dentine may be stained and traced in preparations. This would explain the supposed existence of nerve fibrils in this area. The deduction from silver-stained preparations that microscopically visible nerve fibrils accompany the odontoblastic processes in the tubules would often seem to be based on a misinterpretation of the picture presented by what are, in fact, decomposed remnants of the odontoblastic processes. There is still, however, the possibility that submicroscopic nerve fibrils exist in some of the dentinal tubules,[3] which then would not primarily be expected to contain odontoblastic processes as well. An electron microscopic investigation of this problem did not reveal any submicroscopic nerve fibrils in the dentine,[1] but the material did not comprise all the dentinal tubules of a tooth. In another investigation[3] it was stated that fine nerve fibrils exist in the tubules of the mineralized dentine. The problem still does not seem to be definitely settled.

SUMMARY

1. Nerve fibrils may exist in large numbers in the predentine zone of teeth of persons aged 50 to 70 years.

2. This condition may be pathologic and lead to pictures similar to damaged and regenerating peripheral nerve fibrils.

3. The claviform or fusiform swellings of nerve fibrils in the predentine may be due to degeneration or regeneration and may simulate nerve endings.

4. From the facts that (a) nerve fibrils do not exist at all in the vicinity of the predentine at an embryonic stage, (b) they exist but occasionally in this

tissue in young persons and, (c) they exist abundantly in the secondary predentine of older persons, it is deduced that the enclosure of the nerve fibrils in the predentine is due to advancing years, pathologic conditions, or both.

5. The diversity of opinion concerning the innervation of the teeth may thus be partly ascribed to a tendency to draw general conclusions from too little material and to disregard the dynamics of the tooth.

The photomicrographs accompanying this article represent the right lower premolar of a 63-year-old man.

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Erratum

In the article by Chaudhry, Gorlin, and Mitchell, entitled "Papillary Cystadenoma of Minor Salivary Gland Origin," which began on page 452 of the April, 1960, issue, the last sentence on page 454 should read as follows:

> The data do indicate, however, that the tumor has a predilection for the palate, although other locations in the oral cavity are not immune to its occurrence.

Also, the case reported by Castigliano and Gold occurred in a male patient and not in a female patient as indicated in item 5 of Table I.

Section by the

ARMED FORCES AND PUBLIC HEALTH SERVICE

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PERIADENITIS MUCOSA NECROTICA RECURRENS

*William C. Hurt, Major, DC, USA**

A SURVEY of the literature concerning "aphthous" lesions will reveal an imbroglio of conditions. Included under this heading one may find practically all recorded pathologic lesions that would result in an ulcer of the oral mucosa. Out of this a maze of material, there are certain entities which are seen with more or less regularity in the offices of dentists and physicians. One of the more frequently misdiagnosed of these is that which Sutton[1] has called periadenitis mucosa necrotica recurrens. This condition has also been referred to as aphthae resistentiae (in Europe), Mikulicz's aphthae, stomatitis neurotica chronica,[2] ulcus neuroticum mucosae oris,[3] and recurrent scarifying ulcerative stomatitis.[4] Although the disease is generally considered to be rare, it is becoming evident that careful diagnostic procedures in the examination of oral ulcers disclose more and more persons suffering from this malady.

Periadenitis mucosa necrotica recurrens is a descriptive dermatologic term denoting a recurrent manifestation of deep ulcers of the oral cavity. The condition is frequently confused with the recurrent aphthae commonly referred to as recurrent herpetic stomatitis. It is necessary that the two disorders be differentiated, inasmuch as one is much more serious in its implication than the other.

Sutton,[5] in giving the historical background relating to these chronic ulcers, states:

> Chronic aphthae of this type (ulcus neuroticum mucosae oris, or Mikulicz's aphthae) were described by Sibley (Brit. Med. J. 1: 900, 1899), who thought them "neurotic" in origin, and he quoted Jacobi (1894) as having reported 3 cases and reviewed others. Court (Brit. Med. J. 1: 1214, 1899) wrote of a woman whose lesions were painful, pea to sixpence-size ulcers, which lasted for from 3 to 6 weeks and recurred over a period of 10 years or so. Loblowitz (Arch. fur Derm. und Syphilis 102: 191, 1910) reported examples of this disorder, and Sutton (J. Cut. Dis. 29: 65, 1911) independently investigated and described a case and gave it the name I use. . . .

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Two points in the medical histories of these patients are quite revealing. One is that for years excruciatingly painful ulcers have occurred in the mouth and that a lesion in one stage or another is practically always present. Second, it takes from three to six weeks for the ulcers to heal completely. Besides these two important elements in the history, the patient frequently indicates that he has submitted to many and varied therapeutic measures in an effort to gain relief.

Clinically, these patients may be emaciated, the weight loss having taken place over a period of years as a result of the severe pain experienced upon ingesting food. The actual lesion of periadenitis mucosa necrotica recurrens initially is a small white or yellow plaque or nodule that forms on areas of the oral mucosa which contain minor salivary glands. This plaque increases in size and within a few days there is a sloughing of a central necrotic tissue mass, leaving a deep craterous ulcer. The base of the crater is covered with a whitish fibrinous membrane, while the crater's border is elevated and rolled (Figs. 1 and 2). There is a dusky red inflammatory halo about the circular lesion, which lacks the fiery quality of the halo seen about recurrent herpetic lesions. There may be one or more of these ulcers in the mouth at one time, each in a different stage of healing (Figs. 2 and 3). Careful examination of the oral mucosa will reveal areas of scarring which have resulted from previous episodes of ulceration. This characteristic of scarring upon healing is an important diagnostic point. It has been observed that certain of these patients have suffered so intensely and so constantly with this disorder that they are on the verge of developing psychiatric problems.[6]

In the differentiation of these lesions from herpetic lesions, several things are significant.[7] First, the herpetic lesion heals in from ten days to two weeks; periadenitis mucosa necrotica recurrens heals in three to six weeks. Second, the herpetic lesions are more superficial and do not heal with scarring. Third, the herpetic lesion is generally considered to start as a brief, transient vesicle of the oral mucosa, whereas periadenitis mucosa necrotica recurrens begins as a plaque or nodule.

The condition seems to affect all ages. In three cases that have come under our surveillance during the past twenty-four months, one of which is reported here, there is nothing to indicate an age incidence. One patient was a 20-year-old white woman, employed as a dietitian, who had been troubled for about three years. Another was a 20-year-old white soldier who had suffered from the disorder since childhood. The third patient was a 43-year-old housewife who gave a history of severe ulcers for three or four years. The disease seems to affect the white race predominantly.

Histopathologic examination of one of these ulcers which was excised in toto revealed a small central ulceration in the oral mucosa surrounded by an acute inflammatory reaction (Fig. 4). The epithelium on either side of the ulcer showed areas of slight parakeratinization and an absence of a granular layer. The prickle-cell layer was slightly acanthotic and showed varying degrees of intercellular and intracellular edema, the latter being especially prominent near the ulcer. Scattered throughout the prickle-cell layer, even at a distance from

Fig. 1.

Fig. 2.

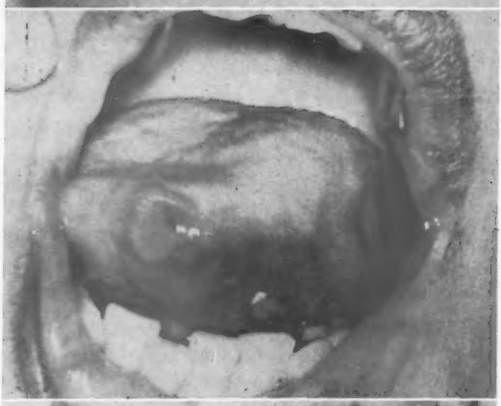

Fig. 3.

Fig. 1.—Large ulcer of the mucous membrane involving the lower lip of a 20-year-old white woman.

Fig. 2.—Multiple ulcers involving the tongue and inner aspect of the upper lip in a 43-year-old housewife.

Fig. 3.—Lip lesion of the patient seen in Fig. 2.

the central lesion, there was an infiltration of polymorphonuclear neutrophils, this being a striking feature of the section (Fig. 5). Distal to the ulcer the basal-cell layer was distinct, being made up of the usual cuboidal and columnar cells. Near the area of ulceration, the basal-cell layer took less basophilic stain and tended to be less distinct; however, it appeared intact. The rete pegs

Fig. 4.

Fig. 5.

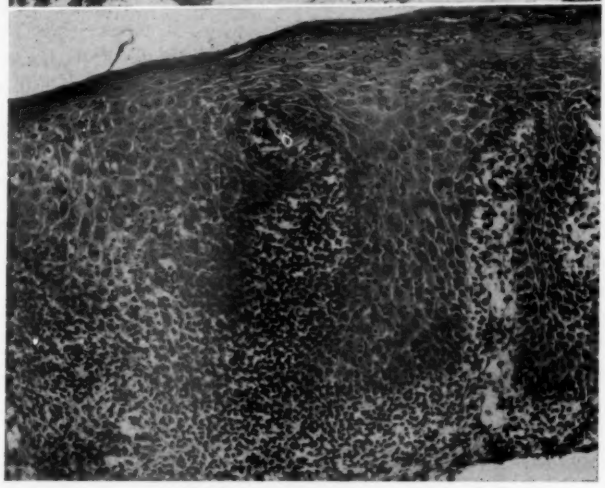

Fig. 4.—Border of ulcer. Note leukocytic infiltration of the marginal and distal epithelium. (A. F. I. P. negative 594562. Hematoxylin and eosin stain. Magnification, ×100; reduced ⅙.)

Fig. 5.—Intense inflammatory reaction at some distance from the ulcer. Note polymorphonuclear neutrophilic infiltration of the epithelium and evidence of "clubbing" of the rete pegs. (A. F. I. P. negative 594563. Hematoxylin and eosin stain. Magnification, ×145; reduced ⅙.)

throughout the section appeared slightly elongated and club shaped. The connective tissue papillae were long and edematous, and contained dilated blood vessels, many polymorphonuclear neutrophils, and some lymphocytes, plasma cells, and macrophages. The inflammatory exudate increased significantly in the vicinity of the ulcer but was seen throughout the entire section (which was approximately 6 to 7 mm. long, the ulcer being about 2 mm. in diameter). Throughout the submucosa there was evidence of edema and dilated blood vessels with perivascular inflammation.

The ulcer itself had a slightly elevated border of epithelium which showed evidence of degenerative change. The floor of the crater was covered with fibrin, which was densely infiltrated with polymorphonuclear neutrophils. Beneath this there was collagenous tissue whose fibers were separated by an intense inflammatory exudate containing some lymphocytes, plasma cells, macrophages, and a predominance of polymorphonuclear neutrophils. There was no evidence of inclusion bodies or fungus.

Treatment of this condition has so far proved unsatisfactory. As with other conditions of unknown etiology, empirical therapy is haphazard and unpredictable. Cortisone and ACTH have been found to give palliative relief in some instances.[7] Repeated smallpox vaccination has been helpful in a few reported cases.[8] A recent preliminary report[4] has indicated that local treatment with triamcinolone acetonide in an adhesive vehicle may be of value in aborting the lesion at its inception and in increasing the rate of healing of well-developed lesions. Para-aminobenzoic acid has been used in some cases in which cortisone has proved of no value; however, when it is used the possibility of liver damage must always be considered.[6] Weichselbaum and Derbes[8] were able to give their patients more relief with prednisone (Meticorten) than with prednisolone or cortisone. In our own cases, we have used prednisone or prednisolone, 5 to 10 mg. three times daily in gradually decreasing doses, for a few days. This reduces the severity of the lesions when patients are in great difficulty, and for a while the patients are free of the lesions. Inasmuch as there are certain medical contraindications to this therapy, we have always sought medical consultations before treating these patients. In one of our patients repeated smallpox vaccination has been of questionable value. It should be pointed out that none of these remedies prevents a recurrence of the lesions, but sometimes therapy helps reduce the severity of the condition to a point where the patient finds it tolerable. Until the etiological factor is identified, there will probably be no adequate method of treatment.

CASE REPORT

A 21-year-old, well-nourished and well-developed white soldier was referred to the Womack Army Hospital Dental Clinic for evaluation and treatment of oral ulcers. He stated that he had suffered from these recurring ulcers for as long as he could remember. Until he was 16 years old he had received no specific treatment for the condition, but at that time he was taken to the University of Michigan Hospital where he was given medication. This relieved his malady temporarily, but when the medication ceased the lesions returned with the same severity as before. (Investigation disclosed that the medication was cortisone.)

The patient grew up in Michigan and stated that he had had all of the childhood diseases. At the age of 9 months, he had contracted mumps and measles, one tending to overlap the other. Following this, he had developed pneumonia. He had nosebleed when working in areas of high temperature. He had never been hospitalized.

Family history revealed that the patient's mother and father were living. The mother had severe hypertension, but the father was apparently in good health. Although both mother and father had an occasional "canker" sore, neither had the severe recurrent lesions. No siblings were affected with oral ulcers, and all were in good health. The patient's 8-month-old boy was healthy and exhibited no evidence of his father's condition.

Clinically, the patient appeared to be in good health. Examination of the mouth at any time revealed one or more extremely painful, ovoid ulcers. These were typically deep, with a rolled border, and they involved gland-bearing areas of the oral mucosa. The ulcers had a dull, inflammatory halo. There were many areas of scarring in the mouth.

The patient made several observations of his own relative to his condition. He felt that the ulcers improved when he was placed in a situation in which an irregular, rushed schedule necessitated poor eating habits. This was noted first while he was in college and working during his off hours. After he was inducted into the Army from college, he developed his most severe episode. He felt that this was due to getting three regular meals a day and having a routine schedule. When he added off-campus university courses to his Army duties, his schedule again became hectic and he missed meals with regularity. His condition then improved. However, when he returned to his home on leave, gained rest, and ate regularly, he suffered severe exacerbations.

Treatment of this patient consisted of prednisolone, 10 mg. three times daily for two days, to relieve the acute phase. He was also vaccinated weekly for eleven weeks. The lesions continued to occur, but the patient stated that they were less numerous and had lessened considerably in severity while he was receiving the series of vaccinations. Six months later, he felt better and was pleased with the result of therapy. (It is important to point out here, however, that at times this patient had experienced partial remissions in the severity of the lesions, and the treatment he received may not have significantly altered the course.)

SUMMARY

Periadenitis mucosa necrotica recurrens is a condition that should be recognized by both physicians and dentists. Although it is frequently confused with recurrent herpes, there are certain factors that aid in differential diagnosis. Treatment of periadenitis mucosa necrotica recurrens is helpful but not curative, and this will probably be the case until the etiology of the malady is discovered.

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THE ISOLATION AND CULTIVATION OF A FILTERABLE FORM RESEMBLING PLEUROPNEUMONIA-LIKE ORGANISMS FROM THE HUMAN DENTAL PULP

Report of a Case

Michael F. Barile, Ph.D., and Abe Sheingorn, D.D.S.,** Washington, D. C.*

PLEUROPNEUMONIA-LIKE organisms (PPLO) are filterable agents grouped on the basis of similar cultural and morphologic properties[1] and placed between bacteria and viruses for convenience until their status is properly established in the microbiologic system of nomenclature.[2] PPLO are similar to viruses in size (150 to 250 mμ),[3-5] filterability,[6] resistance to sulfonamides[7] and penicillin,[8, 9] neutralization with specific antisera,[10] multiplication in host cells (intracellular parasites),[11] and growth preference for metabolizing cells.[12] Unlike viruses, PPLO are propagated on enriched cell-free media under suitable environmental conditions.[13]

PPLO have been isolated from human and animal tissues, including tissues from the urogenital,[14] respiratory,[15, 16] circulatory,[17] articular,[18] central nervous,[19] and alimentary systems.[11, 20] They have been recovered from the saliva[21] and are commonly found in the gingival crevices[22] of normal asymptomatic subjects. Filterable forms have not been previously reported from the pulp.

In this article we will describe a case of acute serous pulpitis from which a pure culture of a filterable form was isolated. We will present a brief discussion of the general nature and properties of PPLO, their relationship to L forms of bacteria, and the possible significance of filterable forms in dental pathology.

MATERIALS AND METHODS

Microbiology.—Specimens of the pulp were obtained and inoculated into PPLO broth and upon PPLO agar[13] for the isolation of both PPLO and bacteria. The cultures were incubated at 37° C. in anaerobic jars with a commercial

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mixture of 95 per cent nitrogen and 5 per cent carbon dioxide and observed after ninety-six hours. Duplicate plates were incubated aerobically at 37° C. Saliva specimens were treated in the same manner.

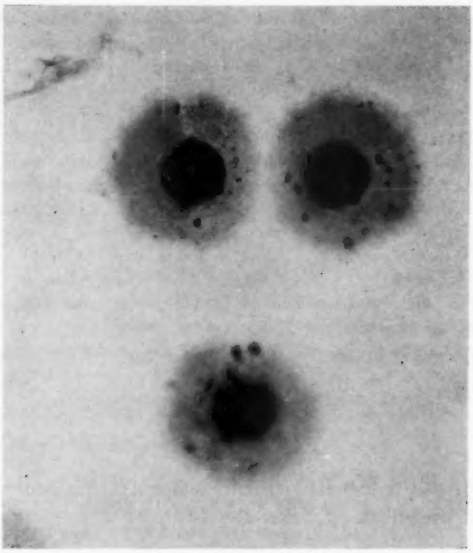

Fig. 1.—Colonies of PPLO isolated from patient with acute pulpitis. (Dienes methylene blue-azure stain. Magnification, ×180; reduced ⅙.)

Fig. 2.

CASE REPORT

M. B., a 34-year-old man, was seen for endodontic treatment of the lower right first molar. The tooth had a zinc oxide-eugenol filling. Previous attempts to relieve the painful symptoms by removal of a shallow amalgam restoration (Fig. 2) plus the oral administration

of 500 mg. of terramycin four times daily for five days were not successful. The pain remained continuous and well localized and it was markedly increased by the application of ice or pressure.

When the zinc oxide-eugenol filling was removed, the dentine appeared noncarious. There was no apparent pulp exposure. A rubber dam was placed on the tooth, and the pulp was exposed under aseptic conditions. The pulp bled freely and showed no gross evidence of suppuration. From a specimen of pulp taken at this time, a pure culture of an organism resembling PPLO was isolated. No bacteria was found in the cultures. The colonies of the organism were typical of PPLO (Fig. 1) and were characterized by (1) a discrete central button with peripheral growth (the "fried-egg" appearance), (2) submerged growth in agar (the colonies were nonscrapable), (3) small size (150 to 175 μ), and (4) a positive reaction to the Dienes methylene blue-azure stain test procedure. The organism passed through a bacterial retaining filter, Type Code HA, with an average pore size of 450 ± 20 mμ (Millipore Filter Corporation, Bedford, Massachusetts).

Although PPLO are identified by their colonial morphology, microscopic examination helps to confirm the identification by ruling out bacteria or fungi. Microscopically, the organisms were typical of PPLO. They appeared as plastic, fragile, gram-negative protoplasmic-like substances with a suggestion of various forms but lacking the discrete morphology characteristic of bacteria and fungi.

When two subsequent cultures over a period of two weeks were negative for PPLO, the root canals were filled in a routine manner. Repeated cultures of the saliva were negative for PPLO but revealed great numbers of other microorganisms.

DISCUSSION

A pure culture of a filterable form demonstrating morphologic properties typical of PPLO was isolated from the pulp of a patient with acute serous pulpitis.

Although PPLO are easily differentiated from bacteria, some of them have properties identical with the "stable" L forms of bacteria (L forms which have lost their ability to transform to their respective bacterial variants). In fact, the colonial and microscopic morphology of some stable L forms is indistinguishable from that of PPLO. Since identification is based on morphology, differentiation of the two forms can be extremely difficult. For this reason, the organism isolated from the pulp may be an L form of bacteria.

The L forms of bacteria differ from PPLO as follows: The filterable L form is genetically related to a particular bacterium. The L form and bacterium may appear individually or together, they may transform from one type to another (with numerous intermediate stages) in a cyclic manner (either spontaneously or by induction), and they may have common antigens. The unstable L forms (the intermediate transformation forms) can be strikingly different from PPLO. During transformation, the intermediate L forms tend to demonstrate morphologic properties characteristic of their bacterial variant.

L forms are identified, therefore, after relationship has been established to a particular bacterium by (1) observations of transformation and/or (2) antigenic cross-reactivity. In contrast, all attempts to relate PPLO and bacteria have failed. For these reasons, most investigators consider PPLO and L forms distinct and separate microbial entities. Other hypotheses exist, however. For example, a PPLO may be considered a stable L form of bacteria which has not as yet been shown or proved to be related to a particular bacterium.

The organism under discussion is considered a PPLO, since it has not shown those properties consistent with L forms.

The isolation of a filterable agent from the dental pulp presents a number of practical and theoretical considerations and suggests a necessity for further investigation to ascertain the significance, if any, of PPLO or L forms in dental pathology. For this reason, investigations designed to establish the incidence of these organisms in acute pulpitis are being continued. Since PPLO and L forms defy cultivation on standard artificial media used in endodontics and do not produce turbid growth, one must emphasize (1) the inadvisability of concluding that "clear-looking cultures" categorically indicate sterility in root canal microbiology and (2) the importance of adequate conditions for growth. These organisms require a high pH (7.6 to 8.0), enriching substances (10 to 15 per cent sera), growth factors (1 per cent yeast extract), and an anaerobic atmosphere (5 per cent carbon dioxide and 95 per cent nitrogen) for growth,[13] and they cannot be identified without the aid of a microscope because of their small colony size (50 to 300 μ) and subsurface growth. Even plates containing numerous PPLO or L forms may give the appearance of "sterile plates" when observed with the naked eye. Consequently, if PPLO and L forms are to be considered in dental pathology, suitable conditions are required for growth, isolation, and identification.

L forms are induced in vitro by exposing bacteria to various agents.[1] Antibiotics, particularly penicillin,[1] are commonly used, and the L forms induced are characteristically resistant to the antibiotic used. This mechanism of transformation affords the bacterium another means of acquiring resistance to antibiotics, and for this reason the significance and possible occurrence of transformation in vivo[1] has received serious attention, particularly in conditions characterized by relapse, recurrence, and antibiotic failures.[23] These circumstances might be explained as follows: During antibiotic therapy, the exciting bacterium transforms into an L form. Since the L form is nonpathogenic[1, 12] but resistant to the antibiotic, it replaces the bacterium and remains dormant, possibly intracellularly, in the host. When conditions become suitable for bacterial reversion, the pathogenic variant may appear manifested by a recurrence of the infectious condition. Continued use of the antibiotics might cause the cycle to be repeated. If the hypothesis presented is valid, a re-evaluation of the use of antibiotics as root canal medicaments is indicated. The antibiotic intended to control and destroy bacteria may inadvertently induce a more refractory agent.

Either agent, the PPLO or the L form (0.15 to 0.3 μ in diameter), could easily be accommodated in the dentinal tubule (1 to 4 μ in diameter) or exist intracellularly in the odontoblastic fiber which fills the tubule. Since the odontoblastic fiber is an extension of a pulpal cell (odontoblast), a filterable form in the fiber would have direct access to the pulp. This would suggest that the point at which a filterable agent can invade the pulp from the enamel is at the dentinoenamel junction where the odontoblastic fiber is first encountered, rather than at the dentinopulpal junction.

SUMMARY

A case of acute serous pulpitis from which a pure culture of PPLO was isolated is presented, and its possible effect on our present concepts of dental pathology and therapy is discussed.

Although no general conclusions are attempted on the basis of one case report, we hope that this report will generate and stimulate further interest in the possible role of filterable forms in dental pathology.

The authors are grateful to Mr. Donald B. Riggs for technical assistance.

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ANNOUNCEMENTS

American Academy of Periodontology

The American Academy of Periodontology will hold its forty-sixth annual meeting at the Miramar Hotel in Santa Monica, California, Oct. 13 to 15, 1960. The theme of the scientific program is "The Role of the Tooth in Periodontal Health and Disease." Any member of the American Dental Association or of a foreign dental association may attend the meeting upon payment of a modest registration fee.

American Academy of Oral Pathology

The Academy held its annual meeting and slide seminar at Northwestern University on April 27, 28, and 29, 1960. The following officers were elected for the coming year: *President,* Robert A. Colby; *President-Elect,* David Mitchell; *Vice-President,* Carl Witkop; *Secretary,* Robert Gorlin.

Pacific Coast Workshop

The second annual Pacific Coast Workshop in Oral Pathology is to be held at the University of Oregon in Portland starting Sunday, July 10, 1960. The subject of the Workshop will be "Intraosseous Lesions of the Jaws."

American Board of Oral Pathology

The 1960 examination for certification by the American Board of Oral Pathology will be held on Dec. 3 and 4, 1960, at the Baltimore College of Dental Surgery, 618 West Lombard St., Baltimore, Maryland. All inquiries relative to examination and certification should be addressed to Donald A. Kerr, Secretary, The University of Michigan School of Dentistry, Ann Arbor, Michigan.

American Institute of Oral Biology

The seventeenth annual meeting will be held in Palm Springs, California, Oct. 8 to 12, 1960, preceding the meeting of the American Dental Association in Los Angeles. The faculty will consist of eight outstanding speakers:

Benjamin H. Ershoff, Ph.D., Director of the Western Biological Laboratories in Culver City, California, will present interesting material regarding conditioning factors in nutritional disease and unidentified nutritional factors and resistance to stress.

John Field, Ph.D., Associate Dean of the School of Medicine, University of California at Los Angeles, will discuss the development of biomedical sciences with special regard to concepts and theories.

Edgar S. Gordon, M.D., Professor of Medicine, University of Wisconsin, will discuss the timely subjects of fat, carbohydrate, and protein metabolism, metabolism of exercise, and the vascular disease of diabetes, which will correlate well with the other lectures.

Wilton M. Krogman, Ph.D., Director of the Philadelphia Center for Research in Child Growth, will speak on genetics and the dentist; the Philadelphia standards of facial growth; and the role of roentgenographic cephalometry in faciodental changes of later life.

Balint Orban, M.D., D.D.S., will describe tissue structure and the function of the periodontium; experimental occlusal trauma (in monkeys) and its biologic and practical significance; wound healing after various methods of periodontal therapy; and roentgenographic interpretation of the alveolar crest and structure of the alveolar bone.

C. M. Pomerat, M.D., Professor of Cytology at the University of Texas-Medical Branch, will enlarge on the various aspects of his general field of cytology and its importance in dentistry.

Hamilton B. G. Robinson, D.D.S., M.S., Dean of the School of Dentistry, University of Kansas City, will discuss the white lesions of the oral cavity, the diagnosis of cysts of the jaws, and the healing process with special relationship to the oral cavity.

Milton J. Schiffrin, Ph.D., Clinical Assistant Professor in Anesthesiology at the University of Illinois and Associate Director of Clinical Research at Hoffmann-La Roche, Inc., will present the subjects of new drugs and their development; placebo-controlled clinical studies and ethics; psychotherapeutic agents (tranquilizers and sedatives); and the management of pain.

Eighth Annual Roentgenology Workshop

The eighth annual Roentgenology Workshop will be held June 24 to 26, 1960, at Linger Longer Lodge, Priest Lake, Idaho. A list of the clinicians and their subjects follows:

Quentin Royer, D.D.S., Mayo Clinic.
Roentgenographic Appearance of Surgical Lesions of Jaws.
The Oral Manifestations of Systemic Disease.

Cline Fixott, Jr., D.M.D., Dental School, University of Oregon.
Technique of Taking Intraoral Radiographs.

Floyd Jacobson, D.D.S., Dental School, University of Washington.
Diagnosis of Enostosis and Exostosis of the Jaws.

Jerome Zeck, D.D.S., Seattle, Washington.
Stuff and Such.

Malcolm Chipman, D.D.S., Spokane, Washington.
Orthodontia—Third Molars.

In addition to the above, arrangements are being made for a one-hour panel discussion on certain technical phases of roentgenology. The Workshop will end Sunday at noon with a one-hour question-and-answer period.

The only requirement for attendance is a sincere interest in roentgenology. All in attendance will be participants and members of the session. The fee is $40.00. As accommodations at the Lodge are limited, reservations will be accepted on a "first come, first served" basis. Those desiring to attend are urged to contact the secretary, Dr. Dennis W. Kelly, S. 3201 Grand Blvd., Spokane, Washington, immediately.

Humboldt University of Berlin Medical Faculty (Charité)

The celebration of the 250th anniversary of the Charité will be held in Berlin, Nov. 6 to 19, 1960, in connection with the 150th anniversary of Humboldt University. Applications for participation should be directed to the Committee for the Preparation of the 250th Anniversary of the Charité, Berlin N 4, Schumannstrasse 20-21, c/o Dozent Dr. med. habil. Dagobert Müller, secretary of the Committee.

Boston University School of Medicine

The Department of Stomatology, Division of Graduate Studies, Boston University School of Medicine and the Massachusetts Memorial Hospitals, announces the following postgraduate courses:

Occlusal Adjustment. Bernard S. Chaikin, Henry M. Goldman, Gerald M. Kramer, William Pendergast, Jack Bloom, and associates. Sept. 12 to 16, 1960. $200.00.

Enodontics. Herbert Schilder. Sept. 12 to 16, 1960. $175.00.

Periodontal Therapy. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, Jack Bloom, and associates. Nov. 7 to 11, 1960. $175.00.

Fixed Bridge Prosthesis. Leo Talkov, David J. Baraban, and Lloyd Warshauer. Nov. 14 to 18, 1960. $175.00.

Full Denture Prosthesis. Chester Landy. Dec. 12 to 16, 1960. $150.00.

For further information about specific postgraduate courses and for application blank, write to Director of Postgraduate Courses, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Massachusetts.

College of Physicians and Surgeons of San Francisco

The College of Physicians and Surgeons, A School of Dentistry, has announced the establishment of a rotating dental internship with the Department of Dentistry of Mount Zion Hospital and Medical Center of San Francisco. The internship will provide experience in general clinical practice and will give the intern an opportunity to gain experience in the various medical services and the departments of the hospital. At the hospital, the intern will be afforded the opportunity of attending rounds and special clinics, as well as special medical lectures that may be advantageous in his training.

At the School of Dentistry, the intern will take additional didactic courses, participate in the special clinic classes, and gain experience as a teacher in areas of special interest. He will also be permitted to attend selective postgraduate courses given by the School of Dentistry.

The intern will be encouraged to initiate or participate in dental research projects at the School of Dentistry and/or the hospital. He will enjoy status equal to that of the medical interns of the hospital, and the stipend will be $175.00 a month, plus board, room, laundry, and uniforms.

Applicants should write to the Dean of the College of Physicians and Surgeons, A School of Dentistry, 344 Fourteenth St., San Francisco 3, California.

Temple University

An intensive participation course in clinical endodontics will be presented Oct. 31 to Nov. 5, 1960, under the direction of Dr. Leonard Parris, assisted by Drs. Harold Rappaport and Robert Uchin.

The objective of this course is to train the dentist in clinical endodontic procedures. Lectures will be supplemented by visual aids. Closed-circuit television will be used. The postgraduate student will utilize diagnostic procedures, initiate treatment, and carry through to completion endodontic therapy on single-and multirooted teeth of clinic patients. Enrollment is limited to ten. Tuition is $175.00. This course will fulfill the prerequisite requirement for the postgraduate course in surgical endodontics to be presented in March, 1961.

For further information and application, write to Dr. Louis Herman, Director, Postgraduate Studies, Temple University School of Dentistry, 3223 N. Broad St., Philadelphia 40, Pennsylvania.

REVIEWS OF THE LITERATURE

BOOK REVIEWS

Endodontic Practice. Louis I. Grossman, D.D.S., Dr. med. dent., Professor of Oral Medicine, School of Dentistry, University of Pennsylvania. Fifth edition. Philadelphia, 1960, Lea & Febiger. 402 pages, 327 illustrations. Price, $7.50.

Formerly entitled *Root Canal Therapy*, this is the fifth edition of a book first published in 1940. The chapters have been rearranged somewhat from the previous edition. Most chapters have changes in material content which reflect recent additions to our knowledge of various aspects of endodontic practice (for example, effects of high-speed instrumentation on the pulp, changes in instruments, special handling of root canal filling materials and new filling materials, evaluation of successes and failures, new antibiotics, etc.). Approximately eighty new references are cited, principally in the chapters on "Diseases of the Dental Pulp," "Sterilization of the Root Canal-Antibiotics," and "Filling of the Root Canal," while a few references have been dropped. The illustrations, with a few additions and deletions, are essentially the same as in the fourth edition. This book is almost a classic and is recommended for student and practitioner alike.

Edward N. Green.

Oral Anatomy. Harry Sicher, M.D., D.Sc., Professor of Anatomy and Histology, Loyola University School of Dentistry, Chicago College of Dental Surgery, Chicago. St. Louis, 1960, The C. V. Mosby Company. Price, $13.50.

This is the third edition of a book which was well received when it made its initial appearance in 1949. The author, who is known for his investigative ability, has accumulated new information on the growth of the skull and the anatomy and function of the temporomandibular articulation.

This volume presents the personal experiences of the author, an authority in the field. It is recommended to the student and the practitioner.

There are 314 text illustrations, many in color.

Thomas J. Cook.

The Surgical Treatment of Facial Injuries. V. H. Kazanjian, C.M.G., D.M.D., M.D., F.A.C.S., D.Sc.(Hon.), and J. M. Converse, M.D., F.A.C.S. Baltimore, 1959, Williams & Wilkins Company. 1110 pages, 1115 illustrations. Price, $22.00.

This second edition of a well-received book contains six new chapters: "Introduction to Fractures of the Facial Bones," "Fractures of the Fronto-Ethmoidal Region and Traumatic Cerebrospinal Rhinorrhea," "Facial Injuries in Children," "Roentgen Examination of the Facial Bones," "The Transplantation of Tissues," and "Scars of the Face." Over 400 new illustrations have been added.

Dr. Kazanjian is well known to the dental profession because of his vast experience and writings relative to facial injuries of World War I.

A very full discussion is given to fractures of the maxilla and mandible. Both closed methods and operative methods are presented. Good results are most consistently realized by the surgeon who is the master of both methods.

This volume presents the personal experiences of the authors. It is recommended particularly to the young oral surgeon who has hospital emergency department responsibilities. He will not turn in vain to this book for help.

Thomas J Cook.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Fortschritte der Kiefer- und Gesichts-Chirurgie (Advances in Mandibular and Facial Surgery, a Yearbook). Volume V. Edited by Prof. Dr. Karl Schuchardt, Director of the Clinic and Polyclinic for Dental-Oral and Mandibular Diseases, Nordwestdeutsche Kieferklinik, des Universitats-Krankenhauses Eppendorf (Northwest German Mandibular Clinic of the Eppendorf University Hospital). Georg Thieme, Stuttgart. Intercontinental Medical Book Company, New York. 182 illustrations. Price, $28.10.

Prevention and treatment of pain in the mandibular and facial regions, observations and therapeutic methods in cases of lip, mandibular, and palatine clefts, new methods of treating fractures of the mandibular and facial regions. Articles have been contributed by numerous well-known specialists.

Fluoridation: Errors and Omissions in Experimental Trials. Philip R. N. Sutton, D.D.Sc. (Melb.), L.O.S. (Vic.), Senior Research Fellow, Department of Oral Medicine and Surgery, Dental School, University of Melbourne. New York, Cambridge University Press. Price, $1.75.

Bacteriology for Students of Dental Surgery. R. B. Lucas, M.D., M.R.C.P., D.P.H., Professor of Oral Pathology, University of London, Pathologist, Royal Dental Hospital of London, and Ivor R. H. Kramer, M.D.S., L.D.S.R.C.S., Reader in Oral Pathology, University of London, Head of Department of Pathology, Institute of Dental Surgery and Eastman Dental Hospital. Second Edition, with frontispiece in color and 58 text illustrations. Boston, Little, Brown & Company. Price, $6.00.

Internal Medicine in Dental Practice. Leon H. Collins, Jr., A.B., M.D., F.A.C.P., F.C.C.P., Associate in Medicine, School of Medicine, Assistant Professor of Medicine, School of Dentistry, University of Pennsylvania. and Martin P. Crane, B.S., M.D., Sc.D. (Hon.), Chief Physician, The Misericordia Hospital, Philadelphia. Fifth edition. Philadelphia, Lea & Febiger. Price, $8.50.

Anatomy for Students of Dentistry. James Henderson Scott, D.Sc., M.D., L.D.S., Reader in Anatomy for Dental Students, The Queen's University of Belfast, and Andrew Derart Dixon, M.D.S., B.Sc., Ph.D., Lecturer in Anatomy for Dental Students, The University of Manchester, Visiting Associate Professor of Anatomy, State University of Iowa. Edinburgh and London, 1959, E. & S. Livingstone, Ltd. Price, $11.00.

Low Intensity Radium Therapy. Charles L. Martin, M.D., Clinical Professor of Radiology, Southwestern Medical School, University of Texas, and James A. Martin, M.D., Associate Professor of Radiology, Southwestern Medical School, University of Texas. With 20 color plates and 118 black and white illustrations. Boston, Little, Brown & Company.

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PUBLISHED BY
THE C. V. MOSBY COMPANY
ST. LOUIS 3, MO., U.S.A.

Official Publication of

THE NEW ENGLAND SOCIETY OF ORAL SURGEONS
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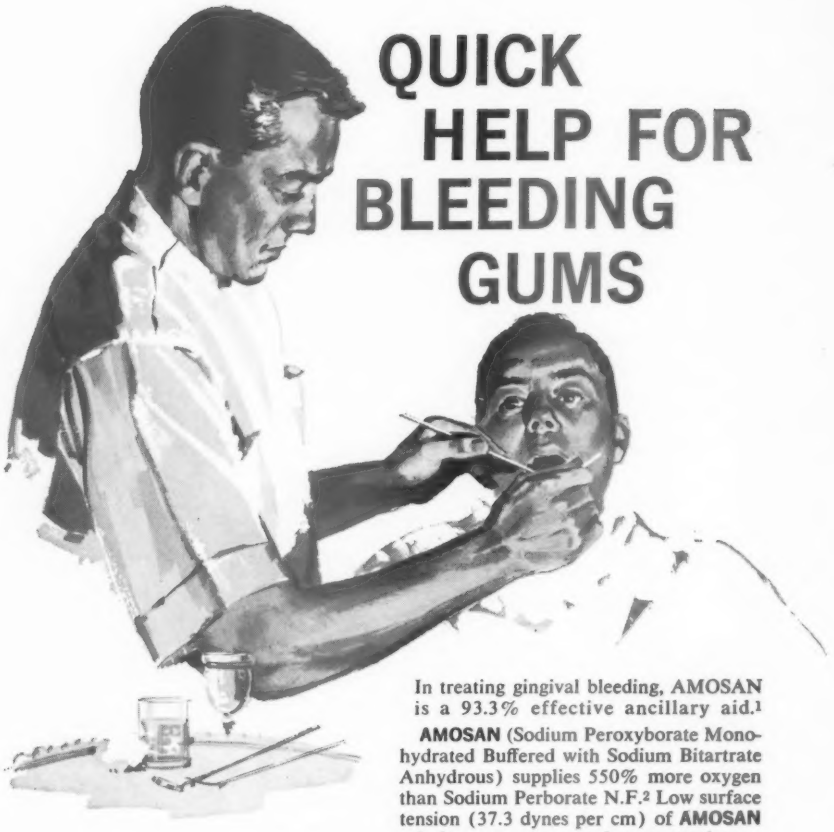

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2. BEHRMAN, S. J.; FATER, S. B.; GRODBERG, D. L.; AN EVALUATION OF OXYGENATING AGENTS IN THE TREATMENT OF GINGIVAL INFLAMMATION. J. DENT. MED., (OCTOBER) 1958.

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ORAL SURGERY
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Vol. 13, No. 6, June, 1960. Oral Surgery, Oral Medicine and Oral Pathology is published monthly by The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Mo. Subscription rates: United States and its Possessions $15.00; Canada, Latin America, and Spain $15.50; Other Countries $16.50. Special rate for dental students and dentists on residency programs, one-half of the domestic rate; for students in other countries, one-half of the domestic rate plus the full amount of the applicable charge for international postage. Single copies $2.50 postpaid. Second-class postage paid at St. Louis, Mo. Printed in the U. S. A.

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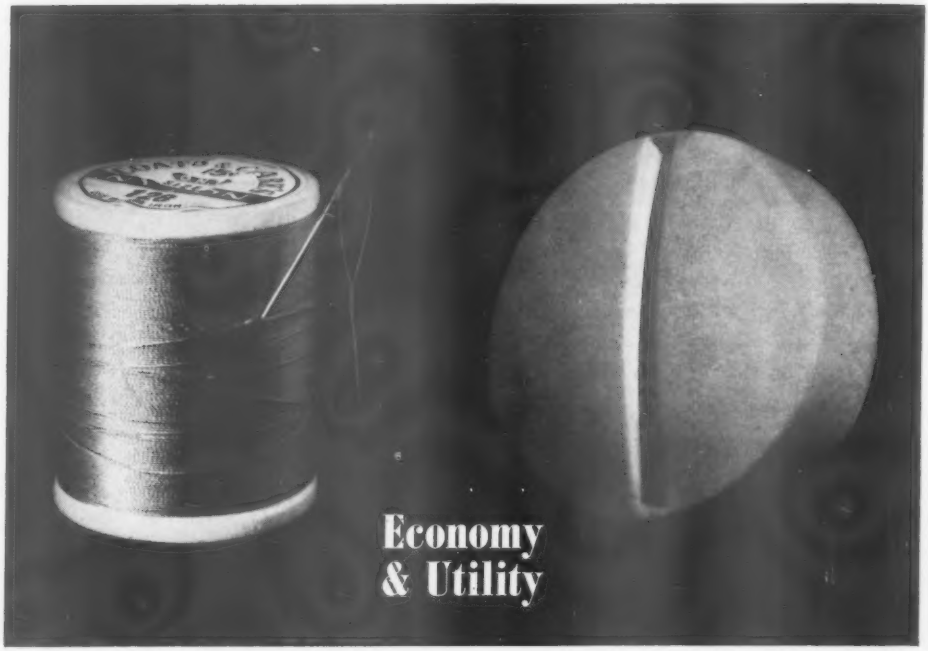

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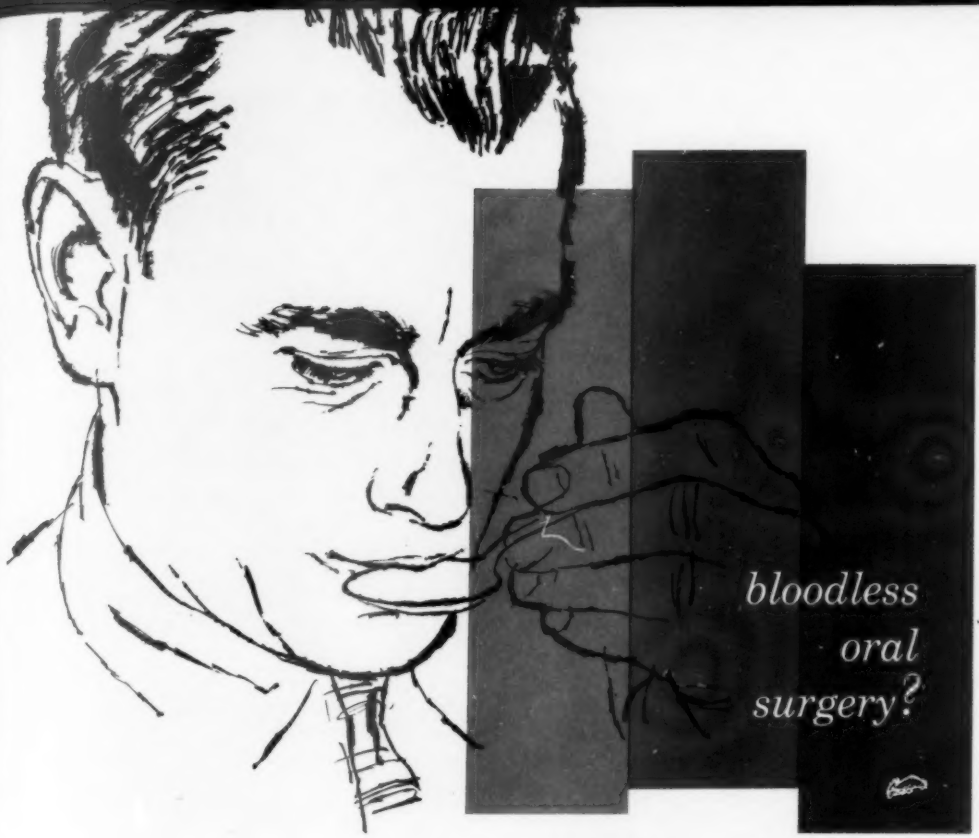

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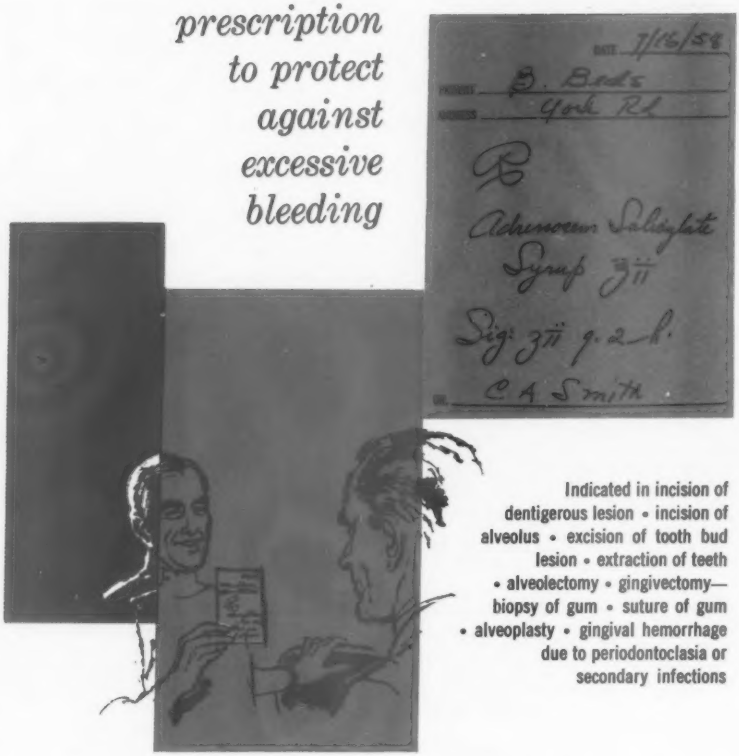

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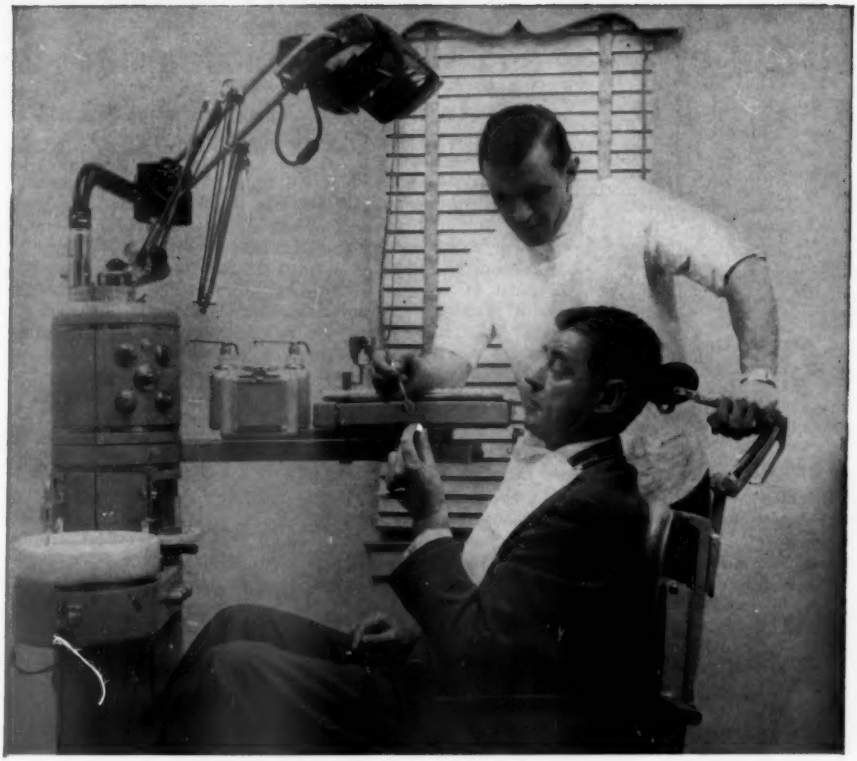

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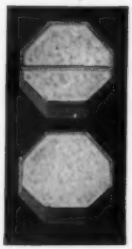

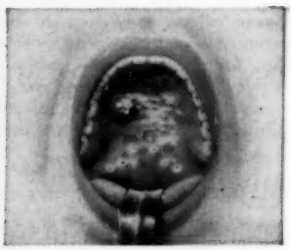

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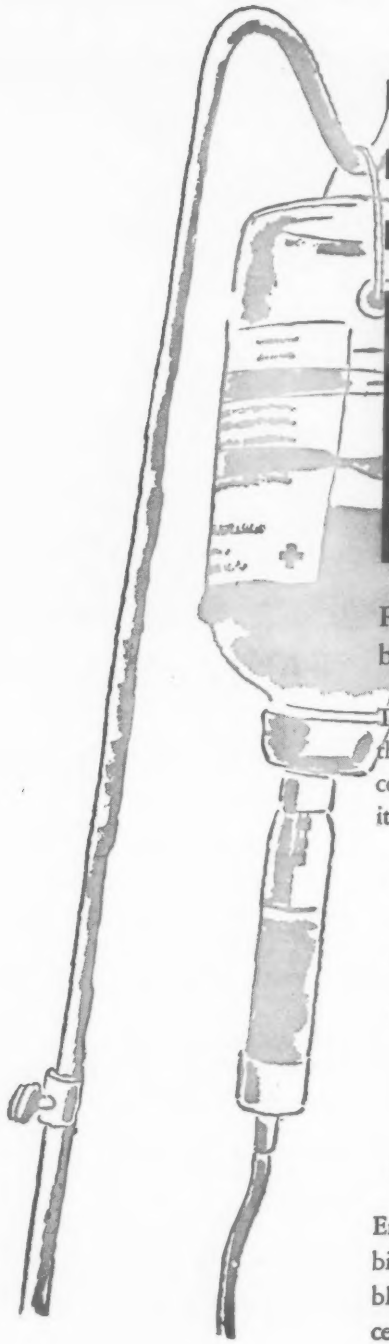

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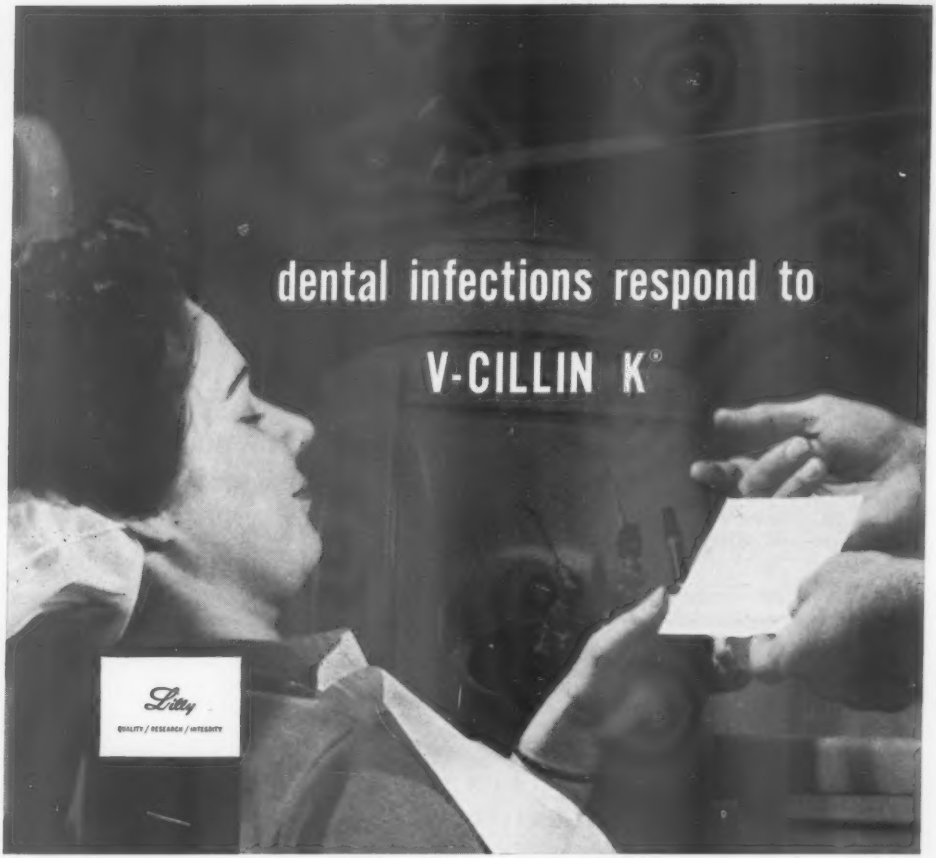

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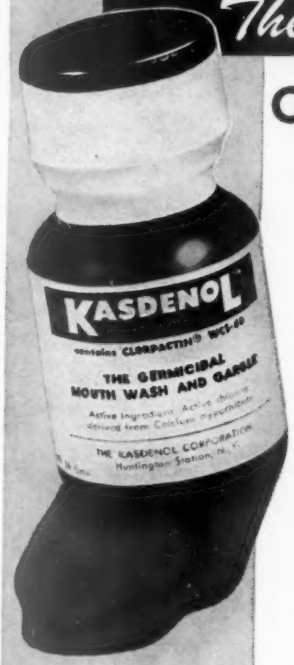

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